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ABSTRACT

This program was organized in response to the rapidly increasing demands placed upon the emergency departments of general hospitals, and in recognition of the fact that the crucial ingredient in emergency department services is physician capability. The training program was implemented for hospital department physicians and other interested licensed physicians, and utilized lecturers recruited from within the State from appropriate professional associations and schools. This publication provides representative papers on the topics presented, with the intent of stimulating the formation of similar training programs in other states. The topics include: resuscitation of the severely injured patient, including restoration of airway, shock, hemorrhage, and fluid replacement; the unconscious patient; pediatric emergency medical care; treatment of burns; cardio-pulmonary resuscitation; cardiac emergencies; psychiatric emergencies; treatment of multi-system injury; and care of simple wounds. (JR)

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Proceedings

New Jersey Training Program for

Physicians in Hospital Emergency Departments

Conducted by

The Medical Society of New Jersey
Committee on Emergency Care

in cooperation with the

New Jersey Department of Health

and the

U. S. Public Health Service
Division of Emergency Health Services

U. S. DEPARTMENT OF HEALTH,
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Introduction

The emergency department of a general hospital is rapidly becoming the most important area in the hospital. Increased use of the emergency service by the general public has created extraordinary demands on the hospital and medical staff to provide better care. No longer can the emergency service be manned by the least experienced member of the medical staff. Unless a physician has had specialized formal or inservice training in emergency medical care, he will be unable to cope with the volume of patients, many of whom are in critical condition, and provide the care that people are now expecting when they appear at hospital emergency departments. The crucial ingredient in emergency department services is physician capability.

A group in New Jersey recognized the need for such training and devoted their time and energy to initiate an action program. The Committee on Emergency Medical Care of the Medical Society of New Jersey recommended the implementation of a training program for hospital emergency department physicians and other interested licensed practitioners.

A questionnaire was sent to each of the 21 component medical society presidents requesting their comments on the training program. All replies were in favor of holding the training and recommended that the program be brought to the physicians.

The Trustees of the Medical Society approved the recommendation of the Committee on Emergency Medical Care and the training program was held at select hospitals and repeated on successive Wednesdays in each of the five judicial districts of the MSNJ. All lecturers were recruited from within the State from appropriate professional associations and schools.

The program was conducted by the Committee on Emergency Care of The Medical Society of New Jersey, in cooperation with the New Jersey Department of Health, and the Division of Emergency Health Services of the U.S. Public Health Service. Other cooperating medical organizations were the New Jersey Trauma Committee of the American College of Surgeons; New Jersey Heart Association; New Jersey Chapter, American Academy of Pediatrics; Council on Mental Health of the Medical Society of New Jersey; and the New Jersey Neuropsychiatric Association.

Sessions were well attended by physicians from all fields of medicine. As a result of this successful venture, Dr. Jack R. Karel, Chairman of the Committee on Emergency Medical Care of the Medical Society of New Jersey, recommends that similar in-service training programs be held annually in every general hospital.

This description of the organization of the New Jersey Emergency Department Seminars, plus representative papers on the topics presented, are provided to stimulate formation of similar training programs in other States.

RESUSCITATION OF THE SEVERELY INJURED PATIENT, INCLUDING
RESTORATION OF AIRWAY, SHOCK, HEMORRHAGE, AND FLUID REPLACEMENT

Eric J. Lazaro, M.D., F.A.C.S. *
Benjamin F. Rush, M.D., F.A.C.S. *
Paul Bolanowski, M.D. *

Survival of the severely injured patient arriving in an emergency room is contingent upon prompt and correct diagnosis and therapy. Life-threatening conditions must be treated first. Treatment is carried out methodically and swiftly. The restoration of impaired ventilation and the control of bleeding are of foremost importance. After this, damage to the central nervous system, abdominal organs, urinary tract and musculoskeletal system is assessed and appropriate treatment instituted.

Respiratory System

Having stopped any external bleeding, preferably by direct compression, attention is directed to the respiratory system. Movement of the chest wall indicates whether the lungs are ventilating. Depressed respirations may occur in a patient with a severe intracranial injury where the problem is likely to be central and not confined to the respiratory organs. Obstruction to the upper respiratory passages may result from extensive damage to the face and mouth and/or retraction of the tongue into the pharynx. A pneumothorax produced by leakage of air through a penetrating wound of the chest wall diminishes ventilatory excursions on that side of the chest. Subcutaneous emphysema over the chest or neck betrays a perforation of the lung or fracture of a bronchus or of the trachea. Finally, fractures of the ribs may impair ventilatory movements.

The therapeutic means available for correcting impaired ventilation include: mouth to mouth respiration; the introduction of an ora-pharyngeal airway; passage of an endotracheal tube; performance of a tracheostomy; endotracheal aspiration of blood and other debris from the tracheo-bronchial tree; introduction of a chest tube or aspirating device to remove air, blood and fluid from the pleural cavity; stabilization of the chest wall when multiple rib fractures are causing a flail chest with paradoxical motion; administration of positive pressure oxygen with the aid of a tight-fitting face mask; and removal of subcutaneous air by multiple incisions in the skin and subcutaneous tissues. Each of these modalities are employed depending on the appropriate need. Assisted ventilation through an endotracheal tube is necessary in an unconscious patient who has difficulty in breathing. The tube may be left in place as long as necessary.

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A tracheostomy is performed if there is obstruction to the respiratory tree above the level at which the tracheostomy tube is inserted. It also helps to reduce the dead space, and aids in tracheal aspiration. Removal of air from the chest is indicated when a pneumothorax has occurred and chest wall excursions are diminished. A pneumothorax could be diagnosed by auscultation but confirmatory X-rays are often necessary. When there is a tension pneumothorax, rapid removal of the air is most important. In the presence of a tension pneumothorax, the chest wound is closed with a compression dressing of vaseline gauze and the intrapleural air aspirated. If air continues to accumulate in the pleural space, a chest tube is introduced and attached to water-seal drainage.

Shock

Shock is difficult to define. In defining shock, clinicians think of it essentially in terms of a rising pulse rate and a drop in blood pressure; the Physiologists regard shock as paralysis of the smooth muscle of the blood vessels with resultant vascular collapse. From the scientific point of view, shock represents inadequate tissue perfusion. While there are different etiological factors in the genesis of shock, it is important to remember that considerable overlap may occur in certain patients. For example, hemorrhage may occur in association with acute myocardial infarction.

In the immediate therapy of impending or existing shock, a catheter is inserted in the cephalic vein to the level of the right atrium. This will assist in the monitoring of central venous pressure and the administration of blood and fluids. Occasionally, in a seriously ill patient, more than one infusion site is obligatory. Blood is obtained for typing and cross-matching prior to the administration of agents that may give rise to serological difficulties that could lead to a transfusion reaction. Subsequent to this, Ringer's lactate solution is administered. If there is no improvement in the central venous pressure and systemic blood pressure after two liters of Ringer's lactate have been given, infusion of cross-matched blood is commenced. Should this not be available, then type O-Rh negative blood may be utilized despite its relative risk. After every unit of blood, sodium bicarbonate is given intravenously to combat acidosis.

When hemorrhagic shock appears to be refractory to therapy, the patient should be examined for conditions that may be responsible for this refractory state. A feeble pulse, muffled heart sounds, stuporousness, semiconsciousness or unconsciousness, are suggestive of cardiac tamponade. Introduction of a large bore needle below the xiphoid into the pericardial sac with aspiration of this blood promptly relieves the tamponade. Hypoxemia and hypercapnea from a pneumothorax could cause hypotension. In an elderly patient with marked pulmonary emphysema auscultatory evidence of pneumothorax may be unreliable and reliance on X-rays becomes imperative. Head injuries, generally, do not produce shock. A flail chest, infection and acute pancreatitis should all be considered in the differential diagnosis of refractory hypotension.

With hemorrhagic shock there is a diminution in the blood volume, fall in venous return, and decreased cardiac output. Various physiologic mechanisms are called into play for compensatory purposes. Capillary blood flow is diminished. The endocrines are stimulated to induce hormonal mechanisms that will restore pressure and eliminate the renal problems involved in controlling blood flow. There are changes in the blood sugar and potassium. Glycogen is removed from the liver. Various biochemical factors such as histamine, serotonin and bradykinin are produced. All of these are attempts on the part of the body to compensate for what has occurred, but may in reality lead to serious consequences in protracted shock.

Organs Affected

Every organ system in the body is affected in shock. There is decreased coronary blood flow. Diminution in the circulation to the liver will produce changes in hepatic adenosine-triphosphate and lactate. The brain is depressed. There is a fall in blood flow to the lungs. It has recently been shown that as a result of circulatory changes in the lungs, blood is shunted away from the pulmonary circulation. If these shunts are significant, (over 50 percent), the prognosis is grave. The organ that is most seriously affected in shock is the kidney. Due to vasoconstriction, renal blood flow is diminished, and redistribution of blood from the cortex to the medulla of the kidney is noted. Renin and angiotensin levels are altered in attempts to improve vascular efficiency. There may be damage to the renal collecting tubules. Pigment is deposited in the tubules and this pigment together with poor perfusion will produce acute renal shutdown. Cellular metabolism is also affected. The cell starts to metabolize glucose anaerobically resulting in an increase in lactic acid. The cell eventually ceases to function as the various intracellular structures are destroyed. Changes in blood volume occur. Arteriolar smooth muscle tension is adversely affected. Increased circulating neuro-humoral agents such as bradykinin, the accumulation of carbon dioxide locally, and, derangements in the muscle of the vessel wall, will all affect the tone of the vessels, resulting in paralysis of the sympathetic system.

Patient Study Methods

It is important to remember that some methods used for studying the patient in shock are generally not available in the average emergency room. Measurement of blood pressure and the pulse rate are still widely employed. These should be recorded as frequently as indicated. A catheter is introduced into the bladder and frequent measurements of urine output determined. If possible, assessment of arterial blood pH, carbon dioxide and oxygen values are helpful. Knowledge of the central venous pressure is necessary in many cases. Observing the status of the skin is still a good clinical point. A cold and clammy skin is usually evidence of shock. The other parameters are less commonly recorded.

Specific Fluid Needs

After assessing the patient's injuries and establishing the various modalities for determining the response to treatment, and having commenced

initial fluid therapy, the specific fluid needs in a particular case are then determined. In addition to recording the blood pressure, pulse rate, central venous pressure, urine output and general clinical state, serial hematocrits are obtained. When recording the central venous pressure certain important points have to be kept in mind. This measures only one particular aspect of cardiac function. Any mechanical defect, such as a kink in the catheter or a pneumothorax, can result in a false reading. A major catastrophe that can occur during central venous pressure determinations is escape of the catheter into the circulation. When this occurs a thoracotomy may be necessary to retrieve the lost catheter.

While blood is often lifesaving, it is important to remember that it is a highly unphysiologic solution. It is cold, has a low pH and contains little calcium but appreciable amounts of citrate. The potassium in "banked blood" is markedly increased. Transfusion reactions are prone to occur. Patients who have liver and renal disease face increased risks. Administration of large volumes of cold blood could result in cardiac arrest or ventricular fibrillation. Blood should, therefore, not be administered randomly without a strong therapeutic indication. If time permits, it should be warmed. In repeated transfusions sodium bicarbonate is given. Administration of intravenous calcium which at one time was thought to be a good therapeutic measure with massive transfusions is now considered injudicious practice. Large amounts of calcium may induce cardiac asystole.

When a patient has been in shock for some time, there is paralysis of the circulatory system and the vascular bed appears to be increased. As a result of this the body seems to require more fluid than is generally needed. For this reason determination of blood volume is helpful, appropriate attention being paid to the particular difficulties that may arise in its assessment. Due to the rapid loss of blood and also to administration of large quantities of blood, the isotopic determination of the blood volume may be inaccurate.

Therapeutic Agents

In addition to blood, the therapeutic agents available for volume expansion in shock include plasma, plasma substitutes and plasma derivatives. Plasma is preferably avoided because of the risk of hepatitis. Plasmanate is an effective plasma substitute that is claimed to be free of viral contamination. For volume expansion, Dextran 70 is preferable to low molecular weight dextran. Other agents employed in shock for specific purposes include sodium bicarbonate, "Tris" or "Than", morphine, oxygen, Ringer's lactate, mannitol, the vasopressors and inotropic drugs. In the treatment of acidosis by sodium bicarbonate, caution must be exercised when the patient has congestive heart failure or renal insufficiency. Small doses of morphine given intravenously are preferable when pain is severe, but narcotic and other depressive agents should be avoided if at all possible. Oxygen is freely administered with one precautionary note--when given for prolonged periods it causes organic changes in the lung structure analogous to that seen in hyaline membrane disease. The exact genesis of this phenomenon is debatable.

Pressor agents like norepinephrine are very seldom necessary and in fact are harmful in hemorrhagic shock. Use of dibenzyline has been found to be useful in the experimental laboratory but evidence to support its efficacy in clinical states is lacking. Dextran 70, which is the high molecular weight dextran, is an excellent volume expander. It is desirable not to administer more than two or three liters at one time to a patient. Excessive amounts of dextran may increase bleeding potential of a patient; the exact mechanism of which is poorly understood. Dextran may also cause difficulties in typing the patient for blood transfusion. In the patient in shock, as was pointed out, there is pigment deposition in the kidneys. When oliguria results from this, mannitol (25 gm in 500 ml of a vehicle) is given rapidly, intravenously. Increase in urine output by this means serves as a test of kidney function to rule out other organic kidney diseases. Mannitol is an osmotic diuretic and it removes the pigment that has accumulated in the renal tubules. "Tris" or "Tham" (Trishydroxymethyl Amino Methane) is a buffer agency which could be employed as a substitute for sodium bicarbonate. In emergency room practice, the need for "Tris" very seldom arises. It is an amino alcohol that is given to correct the acidotic state. The maximum dose is 500 mg. It is most often employed in cardiac surgery but occasionally when massive transfusions are given it may be helpful. However, when respiratory function is depressed, the use of "Tris" may have to be curtailed.

There are very few indications today for norepinephrine and other types of vasopressor therapy. Norepinephrine causes vasoconstriction and though it may temporarily raise the pressure it does not improve tissue perfusion. The specific indication for vasopressors is cardiac arrest, when, after heart action is restored some peripheral vasoconstriction is desirable. For the same reason, when a patient has had an adverse reaction to an anesthetic, a vasopressor may be beneficial. Sometimes Isoproterenol (Isuprel) may be given for shock when a cardiostimulant and vascular effect is desired. A small dose of 1 to 2 mg is administered at a slow rate and the pulse watched carefully. If the patient develops tachycardia the administration of Isuprel should be discontinued.

Position of Patient

What should be the position of the patient in shock? Trendelenberg introduced the "head down" position, not for shock therapy, but for providing better exposure in pelvic surgery. Since World War I, it has been accepted to be a good therapeutic measure as it was believed to improve blood supply to the brain. When one tilts the body in the Trendelenberg position, pulmonary excursions are hampered and vital capacity diminished. The shocked patient is, therefore, preferably kept flat and more blood made available for the brain by raising the legs alone.

Crush Syndrome

Finally, there is an uncommon injury referred to as the "crush syndrome". This condition results from a crushing injury in which extensive local tissue destruction is produced, and extensive extravasation of blood into the soft

tissues occurs. Massive red cell destruction leads to pigment deposition in the kidneys. In such a situation the administration of blood should be withheld until renal function has been assessed. In treating a patient with the "crush syndrome", it is better to administer 1/16 molar lactate and calcium gluconate in the presence of tetany. Potassium and blood are avoided until renal function is restored to normal. The local area is drained and debrided and any infection treated with antibiotics, depending on the nature of the organism. Local dextran, pressure dressings, local surgical drainage, and the management of renal insufficiency are the essential features in the therapy of this condition.

SLIDE I

ORGAN-SYSTEM PRIORITIES IN MANAGING SHOCK

1. Restoration of Airway and Ventilatory Support
2. Control of Hemorrhage
3. Central Nervous System Evaluation
4. Abdominal Examination
5. Urinary Tract
6. Examination of Extremities for Fractures

SLIDE II

SURVEY OF RESPIRATORY FUNCTION

1. Movement of Chest Wall
2. State of Consciousness
3. Face, Mouth and Tongue
4. Penetrating Chest Wound
5. Subcutaneous Emphysema
6. Rib Fractures

SLIDE III

THERAPY OF IMPAIRED VENTILATION

1. Mouth-to-Mouth Respiration
2. Oropharyngeal Airway
3. Endotracheal Intubation
4. Tracheostomy
5. Tracheobronchial Aspiration
6. Chest Tube
7. Stabilization of Chest Wall
8. Positive Pressure Oxygen
9. Incisions for Subcutaneous Emphysema

SLIDE IV

DEFINITION OF SHOCK

1. Physiological - Peripheral Vascular Collapse
2. Clinical - Lowered Arterial Pressure
3. Scientific - Inadequate Tissue Perfusion

SLIDE V

MAJOR TYPES OF SHOCK

1. Hemorrhagic (Blood Loss - Hypovolemia)
2. Traumatic (Usually Hypovolemic with Loss of Blood/Plasma and Extracellular Fluids into Tissue Spaced)
3. Septic (Infection)
4. Cardiogenic (Myocardial Infarction, Arrhythmias)

SLIDE VI

IMMEDIATE THERAPY OF HEMORRHAGIC SHOCK

1. Insert Catheter in Central Vein
2. Type and Cross Match
3. Start I.V. Fluid (Ringer's Lactate)
4. If no Response - Blood
5. Sodium Bicarbonate - 3.75 Gm - Repeat

SLIDE VII

FACTORS CONTRIBUTING TO IRREVERSIBILITY IN SHOCK

1. Hemopericardium
2. Pneumothorax
3. Mediastinal Emphysema
4. Obstructed Airway
5. Intracranial Pathology
6. Concealed Hemorrhage
7. Pulmonary Damage
8. Mediastinitis
9. Flail Chest
10. Sepsis
11. Pancreatitis

SLIDE VIII

ORGAN CHANGES IN SHOCK

1. Heart -- Decreased Coronary Blood Flow
Decreased Cardiac Output
Myocardial Ischemia
2. Liver -- Decreased ATP
Decreased Lactate Breakdown
Decreased Detoxification
3. Brain -- Depressed Sensorium and Leg
4. Lungs -- Decreased Blood Flow
Hypoxia
Carbon Dioxide Retention

SLIDE IX

KIDNEY DAMAGE IN SHOCK

1. Vasoconstriction - Decreased Renal Blood Flow
2. Shunting of Blood from Cortex to Medulla
3. Decreased Glomerular Filtration
4. Decreased Medullary Sodium

5. Loss of Countercurrent Concentrating System
6. Activation of Renin - Angiotensin Mechanism
7. Tubular Necrosis - Pigment Deposition
8. Renal Shut-Down

SLIDE X

CELLULAR CHANGES IN SHOCK

1. Inadequate Perfusion
2. Cellular Anoxia
3. Anaerobic Glucose Metabolism
4. Lactic Acid Accumulation
5. Diminished ATP
6. Energy Functions Lost
7. Permeability Gradients Deteriorate

SLIDE XI

FACTORS AFFECTING ARTERIOLAR SMOOTH MUSCLE TENSION

1. Sympathetic Vasomotor Outflow
2. Circulating Neurohumoral Agents
3. Local Metabolites
4. Blood Gass Tensions
5. Intrinsic Myogenic Properties

SLIDE XII

HEMODYNAMIC AND BIOCHEMICAL MONITORING IN SHOCK

1. Cardiac Output
2. Central Venous Pressure
3. Arterial Pressure
4. Urine Output
5. Peripheral Resistance
6. Arterial Blood pH
7. CO₂ Tension
8. O₂ Tension
9. Lactic Acid
10. Base Deficit

SLIDE XIII

CENTRAL VENOUS PRESSURE

1. Inaccurate Technique - Catheter not in Superior Vena Cava
2. Block of Catheter by Clot, Kink, Valve
3. Mechanical Obstruction of Catheter by Cardiac Tamponade, Mediastinal Compression, Venous Thrombosis
4. Changes in Intrathoracic Pressure - Positive Pressure
5. Intra-Abdominal Pressure when Inferior Vena Cava Employed
6. Hypovolemia may Precipitate Cardiac Decompensation
7. Use Serial Determinations
8. Avoid Catheter Complications

THE UNCONSCIOUS PATIENT

Theodore Goldberg, M.D.*

"The Three B's"

In discussing the problem of the unconscious or comatose patient as he presents in the emergency room, it is not my objective to give you a long list of differential diagnoses that can be obtained in any textbook. Instead, I want to help give you a method of correlating the knowledge that we all have about coma into a working approach to the comatose patient. In doing this, I am going to make use of some simple methods of keeping things straight in our minds. In dealing with a comatose patient, first things must come first, and I like to refer to these as "The Three B's -- Bleeding, Breathing, and Brevity."

When a patient is admitted comatose to the Emergency Room, the first objective is not to determine the cause of the coma. The first objective is to maintain the patient's life. If the patient is bleeding, then provide immediate therapy with Dextran or other colloid; or if that is not on hand, large amounts of saline, until blood is available.

Secondly: breathing. It is a most unfortunate experience to lose a patient admitted with any type of coma because of an inadequate airway. Therefore, whether it is head trauma, or an overdose, regardless of the cause of coma, establishment of an airway must receive primary importance. Too often there is a great deal of ineffective activity around the comatose patient without attention being paid to the first two B factors mentioned previously: bleeding, including the whole problem of shock; and breathing, which is primarily the airway. The third "B" is brevity, and that is just a simple way of telling ourselves to do a brief clinical evaluation of the patient because the baseline findings are important. It is not imperative to do a long series of skull X-rays at 3 o'clock in the morning. It is imperative to do a very brief general physical examination and neurological evaluation in order to have a baseline for further evaluation of the patient. In other words, in addition to the blood pressure and pulse, one should note the presence or absence of cyanosis, the status of the pupils, the status of the chest, and quickly auscultate the heart. A brief examination of the abdomen is of great importance. If the abdomen is soft on admission and becomes rigid later on, it is certainly indicative of an intraperitoneal process, such as bleeding. So, a brief neurological and general evaluation, is indicated; and certainly a long series of X-rays is not indicated. Patients have died on the X-ray table when primary attention should have been given to things such as establishing an airway and treating shock.

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In establishing baselines, valuable time should not be lost. Remember the "Three B's": Bleeding, which refers to bleeding and the general shock problem; Breathing, which is the airway; and Brevity, a brief clinical evaluation. After you have done these primary things for the comatose patient, you are ready to continue.

History

The next point is one which is very important -- obtaining the history. This is a simple thing which sounds ridiculous even to talk about, but it is of overwhelming importance. Do not allow anyone accompanying the patient to leave the emergency room until you have been able to speak to them. Too often, a patient is brought into the emergency room and the policeman or other person with him, seeing that medical treatment is in progress, leaves the emergency room and valuable history is lost. I don't feel that time should be taken to obtain history before you control shock, maintain the airway and perform your initial evaluation. However, at that point do not let anyone who knows anything about the case leave the emergency room. Try to obtain information from these sources directly, and not through a nurse or ward clerk. In terms of medications, every effort should be made to call druggists and doctors to find out as much as possible about the patient.

Specific Treatment

Then the next step is to institute specific treatment rapidly as possible. The comatose state in itself often does not require treatment. Too many times, efforts are wasted in attempting to wake the patient up, when that is not the primary problem at all. In other words, if the vital signs are good, if the airway is maintained, if the state of oxygenation is maintained, then efforts to route the patient usually are not necessary and constitute a waste of time in the therapeutic process. However, other forms of treatment may be important at this point, such as passing a Levine tube and lavaging the stomach.

Jumping to Conclusions

My fifth point is the generalization that we all have a tendency to jump to conclusions in dealing with the comatose patient, and this is a mistake. For instance, the older patient is always thought to have a stroke when he may actually have meningitis, or when he may be the subject of poisoning, or some other unusual form of coma. So don't assume there is a non-treatable form of coma just because of the patient's age or background. Alcoholics are very commonly subjected to such errors in judgement. The presence of alcohol on the breath certainly does not indicate that the patient may not have some other form of coma.

Differential Diagnosis

At this point we come to the differential diagnosis. How can we remember all the different causes of coma? Well, we can do it alphabetically by using the vowels "A E I O U" and the word "TIPS." Everybody has his own way of approaching this problem. This is a plan suggested in a recent medical textbook

and I think it is helpful:

- A - Alcoholism;
 - E - Encephalopathy;
 - I - Insulin;
 - O - Opiates (meaning sedatives of various types);
 - U - Uremia and metabolic causes of coma in general.
-
- T - for Trauma;
 - I - for Infections, such as meningitis and encephalitis;
 - P - for Psychiatric;
 - S - for Syncope (syncope being differentiated from the rest of the causes of coma because we are referring here to syncope secondary to reduced cardiac output.)

We will go into these causes in detail, but this mnemonic should be a help in going through the differential diagnosis. Let's go through them one by one.

Alcoholism

First, as I mentioned before, the presence of alcohol on the breath does not mean that alcoholic intoxication is the cause of the coma. Certainly we all know that alcoholics are especially subject to other causes of coma. The other aspect, the reverse of this, is that the absence of alcohol odor on the breath does not rule out alcohol as the cause of the coma, especially in certain forms of alcohol such as vodka. Also, the patient does not have to look like an alcoholic for alcohol to be the cause of the coma. We are seeing more and more adolescents admitted to the emergency room in stupor and coma, secondary to acute alcohol ingestion. I am sure you are having the same experience. So, because the youngster is an adolescent, and apparently comes from a good background, don't rule out alcohol as the cause of the coma. Alcoholics can develop hypoglycemic reactions just on the basis of the liver disease and chronic alcoholism, so there is no harm done if the coma does not seem to be responding, in giving them 50 cc of 50% glucose. Alcoholics, as you know, are particularly prone to subdural hematomas. Following admission to the emergency room, there is no longer any ingestion of alcohol and it is being metabolized, therefore, the coma should progressively lighten over the next few hours. If it progressively deepens, then one must suspect some other causative factor and, as was mentioned, subdural hematomas are more common in alcoholics than in other persons. Alcoholics also are very much prone to other forms of intoxication, specifically barbiturates, other forms of alcohol such as methanol, isopropyl alcohol, ethylene glycol, etc. So, if the coma deepens instead of lightening, one must suspect either an associated process or another form of intoxication superimposed on the one that you are treating.

Encephalopathy

Encephalopathy is the "E" of our vowel reminders. Epilepsy is generally well recognized. The problem is recognizing the postictal state. Here again, these people are candidates for other causes of coma. Head trauma in the epileptic is common and may result in subdural hematoma. So, if the coma in the postictal state deepens instead of lightening following admission to the emergency room, one must look for other causes of coma. Too, these people have access to large doses of drugs. They are frequently depressed and the ingestion of barbiturates or other drugs may be a causative factor in their coma.

Intracranial hemorrhage, of course, is a serious and common problem. In the traumatic cases, the early neurologic evaluation is of prime importance. Noting the state of consciousness and the state of the pupils and the presence or absence of any lateralizing signs on the very initial brief evaluation can be of major importance in determining the need for emergency therapy. The patient who has intracranial hemorrhage secondary to trauma does not die of the loss of blood, but rather of the increase in intracranial pressure. This may be extremely rapid and the only way that it may be determined that this is going on is a comparison with the initial neurological findings. For instance, if the patient is admitted and the pupils are equal and responsive on admission, and an hour later one pupil is dilated and unresponsive and there is a hemiparesis, then you know that there is rapidly progressive neurologic damage. The patient has not been injured further, so it must be due to a rapid increase in intracranial pressure secondary to bleeding. This should be enough evidence for the neurosurgeon to proceed immediately with burr holes without waiting for extensive X-ray investigation, during which the patient may expire. This is the reason for the very brief early neurologic examination that will provide an excellent baseline for later comparison.

Spontaneous subarachnoid hemorrhage can be one of the most deceptive diagnostic problems. First of all, the classic finding of nuchal rigidity may not occur early at all. It may be a rather late sign. The absence of rigidity on neck flexion does not rule out spontaneous subarachnoid hemorrhage. Also, it may present in a rather peculiar way. I want to cite a case that I think is rather interesting. This is a 35-year-old man, a chemical engineer, who has never had any serious illness. His wife had left him a few months before. He was living alone in a furnished room and had received some psychiatric treatment intermittently. He was found on Saturday morning in the parking lot of a Catholic church. Immediately prior to his collapse he had come out of the church and made an overt pass at a woman passerby and then collapsed in the churchyard. He was brought to the emergency room still conscious, talking in a rather irrational manner. There was no odor of alcohol. He vomited several times and complained of headache. He was rather restless. There was no nuchal rigidity. There were no lateralizing neurologic signs. There were no pupillary changes. He was subjected to vigorous questioning as to the possible ingestion of a toxic agent or alcohol. The police were sent to his room and came back with a small amount of Librium. He claimed he had not taken any of the capsules prior to the incident. Over a period of about 2 hours, a great deal of time was wasted in questioning the man repeatedly about the ingestion

of a toxic substance or suicide attempt. At this point he had a generalized convulsion. A lumbar puncture was done which showed gross blood in the spinal fluid. Subsequently, he developed all the findings of spontaneous subarachnoid hemorrhage. Sometimes the history can be very misleading, and in a young adult complaining of headache and restlessness, one must always suspect spontaneous subarachnoid hemorrhage, regardless of the way it presents and the circumstances under which it presents. Cerebral thrombosis and hemorrhage are usually not different diagnostic problems. The major problem is to make sure the patient is not mislabeled as a "stroke" when there is some other threatening cause.

I am sure I don't have to talk very much with you about encephalitis and mass lesions. We know how these illnesses present. The only thing I would mention is hypertensive encephalopathy which is often a problem in differential diagnosis. These patients almost always have urinary findings and they almost always have eyeground findings consistent with severe hypertension and renal disease.

The lumbar puncture is a very helpful diagnostic maneuver in the comatose patient. If there is any evidence of intracranial pressure, either in the fundi or on the basis of very high initial pressure at the time of the tap, one must proceed with great caution. In general, if there is papilledema the tap should be deferred. If high pressure is discovered only at the time of the tap, then the minimum amount of fluid necessary for diagnosis should be removed.

Insulin

The "I" in our vowel reminders refers to the problems of diabetic coma and hypoglycemic reactions. In preparing this talk, I did an informal poll of the emergency rooms in the area. The people I interviewed all agreed that hypoglycemic reactions were a very common cause of coma or stupor in the emergency room, and that they may present in rather bizarre ways, especially in patients who use the longer acting forms of insulin such as PBI or NPH, and especially in older patients. It is not uncommon for an older diabetic with insulin shock to present as a CVA. I think the general rule should be that if there is any question whatsoever, blood should be quickly drawn, and as soon as that blood is drawn, the patient be given 50 cc. of 50% glucose. As a corollary to this, when using the slower acting insulins, we can't forget that, although we may correct the situation momentarily, the patient is still getting the effect of the insulin. If we simply discharge them without careful direction as far as ingestion of food, they may come back an hour or two later again with a hypoglycemic reaction. I want to emphasize that the classic picture of insulin shock, which is due really to epinephrine release, with sweating, pallor, weakness, syncope, and so forth, is a rapid release phenomenon. The cases that are due to the longer acting forms of insulin often present with a history of bizarre behavior, weakness, and other signs of suggestive of cerebrovascular disease leading to the comatose state. It may be extremely misleading, especially since these reactions may occur on very low doses of daily insulin. I also want to emphasize that any of the oral agents can produce hypoglycemia. Some are more prone than others, such as Diabinese, but any, even Tolinase, can produce hypoglycemic reactions. Again, there is no harm in giving 50 cc. of 50% glucose.

Diabetic coma or precoma usually presents with a very good history. However, the onset of diabetes in a young person may be very misleading. It is not uncommon for a teenager to have the first manifestation of diabetes during the course of a respiratory infection. They present to the emergency room with a history of fever, perhaps a sore throat for several days, dehydration, lethargy, and Kussmaul breathing. These patients can be either in coma or on the verge of very serious coma when admitted to the emergency room with no history whatsoever of pre-existing diabetes. If the initial urine or blood test confirms the presence of diabetic acidosis, then 100 units of insulin should be given immediately, 50 units intravenously and 50 units intramuscularly, because of the delay between the time the patient is seen in the emergency room and admission to the floor. This delay in institution of therapy can be very great and rather critical. An I.V. with normal saline should be started in the emergency room. It is very important to initiate such therapy before the patient is taken to the floor.

Opiates

Opiates - this is the general area of sedatives and tranquilizers. I think these are probably the most common causes of unexplained coma coming to the emergency room. There are really only two conditions which produce a flaccid paralysis and reactive pupils, and these are barbiturate intoxication and hysteria. So, if you see this picture and you don't think the patient is hysterical, think strongly of barbiturate intoxication. The second problem that I would like to alert you to in barbiturate intoxication is the problem of the airway. Laryngospasm is a very serious and sudden complication of barbiturate intoxication, and primary attention must be given to maintenance of the airway. The patient who is admitted with crowing respiration and with a history of barbiturate intoxication is in danger of dying very rapidly unless an airway is put in immediately. Waking the patient is not very important. Supportive treatment is much more important, and there is plenty of time for dialysis and so forth. The important thing is to get whatever is left out of the stomach; and maintain the airway and maintain the fluid intake intravenously. After this is done, consult poison control and whatever other sources are available for specific treatment.

Phenothiazine reactions may present with a picture of Parkinsonian crisis, and this is a little deceptive because frequently the patient is a young person who was brought into the emergency room in a stuporous state, generally rigid, eyes turned upward, drooling from the mouth, in typical Parkinsonian crisis. Unless careful inquiry is made as to the ingestion of phenothiazines, this may be missed or may be attributed to something else such as a CNS process. It is rapidly improved with IV Benadryl 25-50 mgs. The first case that I saw of this was a pregnant woman with nausea and vomiting of pregnancy. She was given Compazine suppositories, and then she began to develop dyskinesia due to the Compazine. She was felt to be having tetany and was given more Compazine to control her vomiting. Then she was felt to be having alkalosis. As a result she was admitted to the hospital in a completely rigid state with a typical Parkinsonian crisis appearance, which was rapidly controlled with IV Benadryl. Caffeine is also effective in these cases. Compazine suppositories seem to be a prime offender, but almost any phenothiazine can do this, and the onset can be very sudden.

Uremia

Uremia refers to the general problem of metabolic causes of coma. You might ask yourself, "What laboratory tests should be done routinely in the comatose patient in an attempt to establish a differential diagnosis?" Certainly a blood count, BUN, blood sugar, electrolytes and urinalysis. I think these would give us a good baseline that will establish most of the causes of metabolic coma. The uremic state may not be diagnosed at all unless you do a BUN. Of course, if the patient is admitted with head trauma which is obvious, you do not need to do the electrolytes or the BUN. We are talking now about the patient in whom the diagnosis of the cause of the coma has not been established. Many of these metabolic causes of coma can be mistaken for other conditions. For example, a myxedematous patient may be thought to have a CVA. The patient with advanced metastatic carcinoma involving the bones may be admitted in hypercalcemic coma. Pulmonary encephalopathy due to respiratory insufficiency and hepatic coma due to liver disease are important and treatable metabolic causes of coma. It is particularly important to recognize the precomatous and comatous state in respiratory disease to initiate emergency measures to improve ventilation. Remember - borderline compensation is status asthmaticus can be converted to coma rapidly by the use of even small amounts of sedation.

Trauma

Now, to use the work TIPS, as I mentioned before. T is for Trauma which may be direct and obvious, or the trauma may be indirect; that is, it may be in itself one of the results of the conditions causing the coma, such as a CVA. Also trauma may precipitate other forms of coma such as Addisonian crisis or diabetic acidosis.

Infection

Infection may be a cause of coma. As a general rule, in any older person who presents with an altered mental state, a lumbar puncture should be done. They may have meningitis; and not be a CVA, and they may not present with classic findings. Their nuchal resistance may be attributed to osteoarthritis. Bacteremia, especially gram negative, may present without the classic picture of an infectious disease. This is especially true in patients who have had previous manipulation which may result in bacteremia, such as prostatic surgery. The elderly patient may present with a personality disturbance, a low-grade fever, and may have bacteremia, which may be completely overlooked.

Acute bacterial endocarditis in the older patient may be present with minimal or no murmurs, personality disturbance, and practically no fever. Unless blood cultures are done, this will never be discovered. It is also not uncommon for pneumonia or other infections, which seem rather obvious, to present with personality disturbances in the older patient, and sometimes even in the younger patient. It is not uncommon for an acute organic mental syndrome to be present in acute infection, such as pneumonia. Several years ago, we admitted a patient with an acute paranoid schizophrenia complete with hallucinations, and this was found to be precipitated by a viral pneumonia. This is not to say that this patient did not have a pre-psychotic personality, but the

stress of this acute illness caused the acute psychosis to develop, and it cleared when the infection cleared.

Psychiatric

Psychiatric diagnosis is of importance mainly because it should be diagnosed only by exclusion. Too often we attribute a bizarre mental state to psychiatric causes, and I would say that if one is judging on which side more mistakes are made, it is more common to mistakenly call a condition psychiatric when it is actually physical, than vice versa.

Syncope

Syncope: due to reduced cardiac output. First of all, a confusional state or depression of mental function may occur in the patient with heart failure, and pre heart failure is the main cause, with the mental changes very much secondary. Again, an organic brain syndrome, or what is also referred to as a toxic psychosis, may be the result of the underlying cardiac disease of poor cardiac output. Also, in the patient who is admitted to the emergency room with persistent loss of consciousness and a history of having a heart attack, there is reason to suspect a different diagnosis unless the patient has some form of arrhythmia at the time of examination. The arrhythmia is the clue to the cardiac cause of the syncope, as in Stokes-Adams Syndrome. Persistent syncope in the cardiac should lead to a search for some other diagnosis. I would point out that spontaneous subarachnoid hemorrhage can have EKG changes simulating myocardial ischemia or infarction.

I would like to close by just saying a word or two about heat syndromes. Basically, in dealing with the comatose patient, there are two types of heat syndromes; the first is heat stroke and the second is heat exhaustion. Heat stroke is the failure of the sweating mechanism, whereby the patient is not able to conserve heat. He may even absorb heat from the ground or surrounding objects. It is more common in alcoholics than in other people. The body temperatures go up to terrifically high levels, 106 to 110 degrees. This patient presents with a sudden onset of coma; the skin is dry, not moist or red, and the patient is deeply comatose. Since the primary problem is the inability to conserve heat, placing the patient in a cold bath is the treatment. Heat exhaustion is a different situation. This is due to excessive loss of fluids via sweating and this usually has a more gradual onset, but rapid. Usually patients are not deeply comatose; they may be confused. Their body temperature is not the same as in the heat-stroked patient; 101 degrees is about the highest, sometimes 102 degrees. These patients respond to both oral and intravenous saline. This is not as common as it once was because of the prophylactic use of salt in people who work in situations where they perspire excessively.

PEDIATRIC EMERGENCY MEDICAL CARE

Theodore Kushnick, M.D. *

Introduction

I have been asked to discuss a number of pediatric emergencies from the aspect of "what to do and how to do it." I shall attempt to follow this directive as closely as possible, but I shall also indicate how one may quickly find out what the emergency is, ie. diagnosis; how severe, ie. prognosis; before doing anything, ie. treatment.

General Immediate Care

The initial care of the severely injured child is essentially that of first aid in an effort to maintain life. The upper airway should immediately be examined to remove obstructions which would interfere with positive pressure breathing or mouth-to-mouth resuscitation, if the latter procedures are necessary for a respiratory arrest. Mouth-to-mouth resuscitation in an infant must be gentle with the nose and mouth covered. The rate is 26 to 30 per minute. In the older child, the nose can be closed and the mouth alone used. Oxygen therapy with positive pressure breathing may be instituted subsequently.

Simultaneously, or as soon as possible, the presence or absence of heart-beat should be determined. If cardiac arrest has occurred, external cardiac massage should be promptly started. The younger the child, the less the pressure required by fingers or thumbs of both hands to compress the mid sternum with sharp vertical thrusts approximately 3 to 4 cm. at 80 times per minute. Blood pressure should be determined and followed while using this technique. Intracardiac adrenalin is frequently resorted to, but the benefits of such therapy are questionable.

The patient should be quickly weighed in order to determine doses for subsequent therapy. If this is impossible, the last known weight should be obtained from the parents, or an accurate estimate made.

All external hemorrhage must be stopped by pressure application. Blood should be withdrawn for crossmatching for transfusions and a cut-down performed for intravenous fluid therapy. In an extreme state of shock, the cut-down vein may be too constricted for cannulation and it is senseless to go on attempting cut-downs at other sites. At that juncture, it is still possible to put the patient in the Trendelenburg position, insert a small needle into the external jugular vein and allow colloid or saline to run in at the rapid rate of 400 ml. per meter square. (A newborn is approximately 0.2 meter sq.; 1 year old approximately 0.5 meter sq.; 3 to 4 years old 0.6 meter sq.; 9 year

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old 1 meter sq.; extrapolations can be made as necessary.) An assistant can hold the needle in position. After one hour of such therapy the patient's blood pressure is usually elevated to a point where cannulation at the original cut-down site can be more readily performed.

The patient's clothes should be removed to permit rapid physical examination. X-ray examinations should be performed as indicated by the physical findings.

An unconscious patient requires gastric aspiration, with gastric contents being saved for subsequent studies if necessary.

Tetanus antitoxin is not required if a patient has had basic immunizations and a previous booster dose. Toxoid therapy can be given instead. If antitoxin is required, 250-500 U. human hyperimmune antitetanus globulin is the preferred treatment.

Burns

The first evaluation must be concerned with the airway. If there are severe facial burns, consider tracheotomy under bronchoscopy before marked swelling of the cervical tissue has occurred.

Intravenous fluid therapy is dependent upon the estimation of depth of the burn and percentage of body surface burned. The usual tendency is to overestimate the damage. The rule of nines can be applied to the varying age groups if there is alteration of allowances for head and lower extremities. The trunk and arms are virtually the same in all ages being 38 to 40% and 16 to 18% respectively. The head of the newborn comprises 18% of the body surface and decreases by approximately 1% per year of age to a 6% adult value at 12 years. The newborn's legs comprise 26% and gain 1% per year of age to an adult total of 38% by 12 years. Fluid therapy is calculated to a maximum of 50% for 2nd or 3rd degree burns. (Scald causes a 2nd degree and flame produces a 3rd degree burn.) The fluids are as follows:

Colloid (e.g. plasma) - only if necessary, ie. if patient is in shock half cc. per kg. per % 2nd or 3rd degree burns.

Normal Saline - $1\frac{1}{2}$ cc. per kg. per % 2nd or 3rd degree burns.

5% Dextrose Solution - to maintenance requirements for age. 125 cc. per kg. for less than 1 year of age to 75 cc. per kg for over 2 years of age.

One-third to one-half of the fluid is given in the first 6 to 8 hours; and one-half to two-thirds of the amount is given in the subsequent 16-18 hour period. These are calculated from the time that the burn occurred, and not from the time of hospital admission. Intravenous saline and colloid are given in one-half their original calculated amounts during the second 24 hour period.

After 24 hours, colloid may be necessary and should consist primarily of plasma for scalds or flame burns up to 30%; and, possibly, whole blood and plasma in a 1 to 2 ratio for flame burns and scalds over 30%.

The best indication for adjusting the administered fluids is the clinical condition of the patient. The parameters to be followed include blood pressure, hematocrit, urine output and urine specific gravity. Urinary catheterization is necessary in most patients. The blood pressure should be maintained above 90 to 100 systolic; hct. in the range of 35%; urine output at 10 to 15 ml. per hour for the infant, to 25 to 35 ml. per hour for the 10 to 12 year old child; urine specific gravity below 1.025. If the patient is oliguric after 12 to 18 hours, he does not have renal shutdown but probably requires more fluid. A test water load of 20 ml/kg of 5% D & W given within 30 to 45 minutes usually produces diuresis and indicates satisfactory renal function.

After initial cleansing of the burn with phisohex followed by washing with sterile water, local care is best handled with sulfamylon ointment or 0.5% silver nitrate soaks. If silver nitrate is used, close monitoring of serum electrolytes on a 4 hourly basis is mandatory and adjustments must be made in the intravenous solutions to compensate for NaCl loss.

COMA AND SEIZURE

Diagnosis

There are numerous causes for coma and seizure and a rapid diagnostic workup is necessary. The two conditions frequently occur in the same patient and have the same etiology. Seizures must be stopped quickly and this treatment will be dealt with subsequently.

It is easiest to consider the most likely diagnoses by age groups:

- 1) Infants - consider meningitis, fluid and electrolyte imbalance, head trauma and subdural hematoma, inborn errors of metabolism, and febrile seizures after the age of 6 months.
- 2) Toddler - consider poisoning, meningitis, head trauma, and febrile seizures.
- 3) Pre-schooler - the same as the toddler, plus the possibility of diabetes and brain tumor.
- 4) 5 to 10 year age range - head trauma, diabetes, epilepsy, glomerulonephritis, and brain tumor.
- 5) Over 10 years - diabetes, epilepsy, coarctation of the aorta.

Physical Examination

General - Primary central nervous system disease causes increased tone in the extremities, increased deep tendon reflexes, and pathological reflexes.

CNS symptoms secondary to systemic disease are more likely to include diminished tone, absent deep tendon reflexes, and lack of focal neurologic signs.

The tone of the extremities can be determined by lifting and dropping the limbs. Measure the vital signs; increased intracranial pressure produces hypertension and bradycardia. Any injured patient with coma and seizures who has decreased blood pressure and increased pulse is bleeding elsewhere than in the CNS.

Notice the respirations; if markedly depressed, poisoning is likely. If respirations are increased and the breath has acetone odor, diabetic coma is probable. If the blood pressure is markedly increased in both systolic and diastolic components, the possibilities include **glomerulonephritis** or lead intoxication.

Head and skin should be examined for signs of trauma. Bulging fontanel in the infant and papilledema in the older child indicates intracranial hypertension. Auditory canals should be examined for blood. Nuchal rigidity should be noted.

Eye Examination - Pupils that are fixed and constricted are indicative of poisoning, such as codeine or phenobarbital; unilateral fixed dilated pupils indicate focal increasing intracranial pressure; dilated fixed pupils bilaterally are a sign of preceding seizure or deep coma; absent corneal reflex and decerebrate posture are signs of mid-brain damage and indicate a poor prognosis. Unequal pupils indicate the possibility of a lesion on the side of the widest pupil. The physician should be alert, however, to the possibility of a child having previous anisocoria (unequal pupils) as a normal variant, which may be confusing. Cycloplegics should not be used because they can interfere with continuing evaluation of **pupillary** signs. Nystagmus is indicative of a posterior fossa lesion. Retinal hemorrhage and papilledema indicate increased intracranial pressure.

Laboratory - Draw blood for FBS, BUN, electrolytes including calcium and phosphorus, blood culture, CBC, and save some blood for future use, eg. lead level, acute viral studies. Catheterize for urine studies and urine output. Lumbar puncture is mandatory for accurate diagnosis if there is absence of head trauma. If there are signs of increased intracranial pressure, use only the CSF that fills the manometer for analysis and then remove the spinal needle.

Therapy - Oxygen should be given as necessary. I.V. fluids are to be given at 75% of maintenance requirements, ie. maintain the patient on the "dry side." Specific therapy for coma can only be given when the diagnosis is established by the initial workups. If the patient shows signs of increased intracranial pressure, the agent of choice is intravenous mannitol 1.5 to 2 gm. per kg. given as a 20% solution in 5% D/W over a period of 10 to 20 minutes. Dexamethasone may be used although its benefits have been questioned. If used, the dose is 10 mg. stat initially, being reduced to 4 mgs. on a q4 to 6 h basis over a period of 48 hours.

Therapy for seizures is similar with regard to oxygenation and fluids. Temperature elevation can be reduced by cool water sponges. In order to stop the convulsions, phenobarbital or amytal may be used in a dosage of 5 to 6 mg. per kg. I.M. or by slow intravenous route. One-half the dose may be repeated in 15 minutes if necessary. For prolonged seizures which are generally associated with previous brain damage, valium (diazepam) may be given undiluted in a dose of 5 to 7 mg. per meter square intravenously to a maximum dose of 10 to 15 mg. per meter square; administered over a period of 5 minutes. 4% of the patients may experience apneic spells with this therapy.

Shock

The cause of shock in most pediatric patients is that due to hemorrhage, burn, or large extra-renal losses of water and requires blood and/or other fluid therapy. For transfusion, 10 cc. of whole blood per pound of body wt. is given, but an actively hemorrhaging patient may require a much greater amount. The latter point can be determined by monitoring the vital signs, especially blood pressure and pulse rate. The rate of infusion should be large initially, as indicated previously. Fresh whole blood is preferred, and blood that is over 5 days of age should not be used. Type O Rh negative blood, serum albumin or normal saline may be used freely in an extreme emergency. The smaller the patient, the more likely he is to experience severe electrolyte imbalance if large amounts of aged blood are used.

The etiology on non-traumatic hemorrhage should be sought, i.e., leukemia, aplastic anemia, idiopathic thrombocytopenic purpura and hemophilia.

Anaphylactic shock - If the injection, sting, etc., is in a limb, immediately apply a tourniquet proximal to the site and inject .25 cc of adrenalin 1:1000 into the site and .25 to .5 cc I.M. in another limb. The patient may require subsequent doses which can be given slowly I.V. with 10 cc saline. Subsequently, the slower acting benadryl or cortisone may be given. Oxygen and positive pressure breathing are required.

Respiratory Distress

Croup - The child with croup has severe inspiratory distress. The predominant etiologies are infections and mechanical obstructions due to foreign body inhalation. The epiglottis must be examined in all cases of croup. If inhaled foreign body is probably by history or by neck and chest X-ray, immediate bronchoscopy should be performed.

In all cases of croup, the patient will require must inhalation, with oxygen for cyanosis, if present. Respiratory depressant therapy must be avoided. If the patient's vital signs show a rising pulse and respiratory rate, and auscultation reveals diminished aeration, tracheostomy under bronchoscopy should be performed.

Intravenous fluids are required.

Asthma - It is important to realize that the main causes of asthmatic deaths are those due to various therapies, be it toxic doses of aminophyllin or adrenal cortical insufficiency secondary to prior prolonged adrenal steroid usage.

Initial therapy is aqueous epinephrine 1:1000 given in a dosage of .2 to .3 cc. subcutaneously. This has short duration effect and aminophyllin may be used with restrictions as to dosage and frequency. The maximum dose of amonophyllin is 3 to 4 mg. per kg. by slow I.V.; 5 mg. per kg. orally; or 7 mg. per kg. per rectum; given at 8 hour intervals for no more than 3 to 4 doses. Antihistamines are worthless and respiratory depressants such as codeine cough syrups are to be condemned. The patient requires I.V. fluids and high humidity tents. Oxygen should be given for cyanosis if present, realizing that any patient who is cyanotic in oxygen has an arterial CO₂ of over 65 mm. of mercury and is on the verge of respiratory failure. The mainstay of intravenous fluid therapy is sodium bicarbonate 1½ to 2½ meq. per kg. given in a 15 to 30 minute period. Such therapy is extremely effective in combatting metabolic acidosis and causing a return of bronchomotor response to epinephrine.

Adrenal steroid therapy is not indicated but many physicians do use it. If it is used, it should be tapered from an initial large dose and discontinued in 2 to 3 days in order to avoid adrenal suppression. One week of treatment causes adrenal suppression for over 1 week to as long as 1 month after discontinuance.

In extreme cases, with resultant respiratory failure, intermittent positive pressure breathing with oxygen via endotracheal tube will be required for periods of 20 to 30 hours. It will be best to admit any asthmatic patient who returns to the emergency room twice for therapy.

Staphylococcus Aureus Pneumonia - This type of pneumonia can be overwhelming and produces empyema and pyopneumothorax. Chest tubes are required for drainage of empyema and air. Antibiotic therapy with bactericidal agents resistant to penicillinase producers is mandatory.

Cardiac Emergencies

A diagnosis of congestive heart failure indicates that the patient's pulse is over 170 to 180 per minute; respirations are elevated, usually in the range of 40 to 50 per minute; liver enlargement is present and over 3 cm. below the right costal margin; neck vein distention is present in the older child; cardiomegaly is apparent on chest X-ray; and the patient has underlying congenital heart disease or old rheumatic heart disease.

Digitalization is not always necessary and should be used with extreme caution for acute myocarditis, be it rheumatic or viral; and in patients with glomerulonephritis.

The dose of digoxin for the newborn is .03 to .05 mg. per kg., I.V. or I.M., with parenteral maintenance being one-tenth to one-fifth of the digital-

izing dose.

From 2 weeks to 2 years of age, the dose is .04 to .06 mg. per kg., I.V. or I.M., and maintenance one-tenth to one-fifth of the digitalizing dose, I.V. or I.M.

For those over 2 years, the dose is .02 to .04 mg. per kg. I.V. or I.M. with maintenance being one-fifth of digitalizing dose parenterally. One-half the digitalizing dose can be given initially followed by a quarter of the dose twice more at 6 hourly intervals. Pulse rate should be determined prior to the 2nd and 3rd doses and if less than 120, the drug should not be given. Rotating tourniquets may be a lifesaving or helpful measure at times using three extremities, changing them every 10 minutes and rotating them.

The Paroxysmal tachycardia causes cardiac failure in the infant. Carotid sinus pressure can stop this phenomenon on occasion as it will in an adult, but it is not as effective and usually digitalization is necessary.

An adjunct in therapy, especially for infants, is the use of ethacrynic acid 1 mg. per kg. I.V. in 5% D & W, given in a period of 5 minutes. This results in a diuresis beginning in 30 minutes and lasting for 3 hours.

Poisonings

General - If the poison does not "stink or burn" induced emesis is the best therapy. Gastric aspiration and lavage with saline may be performed subsequently. If the patient is comatose, previously indicated measures should be performed. Supportive intravenous fluid therapy is necessary and knowledge of the metabolism of the particular poison should be sought, so that specific therapy may be given where indicated. Have the parent bring in the container so that the substance and amount ingested can be estimated. If the patient has ingested an antiemetic, such as phenothiazines, he will not be able to respond to syrup or ipecac emesis and will require gastric aspiration and lavage.

Salicylate Intoxication - If the patient has ingested an estimated 100-150 milligrams per kilogram as a single dose, or has had a chronic salicylate ingestion, it is best to admit him for observation and treatment. If a toxic amount has been ingested and the patient is asymptomatic, give emergency treatment and admit him to the hospital. It may be that he is being evaluated too early in his course to have symptoms. 1) Induce emesis with 15 to 30 cc. of syrup of ipecac followed by a glass of water. 2) Pass gastric tube and aspirate the remaining material. 3) Subsequently, lavage. 4) Start intravenous fluids and alkalinize the patient with sodium bicarbonate $3\frac{1}{2}$ to 5 meq. per kg. given over 2 to 3 hours. If the urine is not alkaline within 4 hours, repeat the course of NaHCO_3 therapy. Some regimens consist of giving 20 cc. undiluted 7½% sodium bicarbonate if the patient weighs less than 10 kg. and 40 cc. if over 10 kg., in a period of 5 to 10 minutes; followed by a continuous I.V. infusion of saline and dextrose at 2 to 3 cc. per minute. 5) All regimens require calcium and potassium intravenously.

Peritoneal dialysis with 5% albumin in electrolyte dialysis solution is indicated if the patient worsens despite I.V. fluids. The urinary bladder should be empty; a No. 16 trocar can be inserted 1 inch below the umbilicus; 60 to 105 cc. per kg. (preferably 75 cc. per kg.) can be administered in 20 minutes; equilibration requires 1 to 2 hrs.; then, gravity drain. This procedure can be repeated 2 to 3 times and PRN. This therapy is extremely effective, especially when combined with simultaneous I.V. sodium bicarbonate.

Exchange transfusion can be considered if there is no improvement after the above therapy and the patient's salicylate level is near the lethal range as determined by Done's nomogram; or if the patient has methyl salicylate poisoning. This measure requires at least twice the patient's estimated blood volume. The latter can be rapidly approximated as being 10% of body weight. Additional therapy should include 1 to 2 mgs of Vitamin K; and intravenous glucose to combat hypoglycemia in the sensitive patients.

As recently demonstrated, the use of activated charcoal is very effective in the initial treatment of salicylate, barbiturate and other intoxications. The activated charcoal binds these agents, when given by mouth. Unfortunately, the dose for salicylate intoxication must be 10 times the estimated amount of salicylate ingested. This leads to practical difficulties in that the ingestion of 3gms. of salicylate will require 30 gms. of activated charcoal administered as a slurry.

Lead Poisoning - General: Multiple diagnostic tests must be performed. There is no one test, including the serum lead level, which is always positive. If one or more of a battery of tests is positive, the patient should be treated for lead intoxication.

Specific: 1) Mannitol - If the patient has severe lead encephalopathy and increased intracranial pressure, use as indicated previously. 2) BAL and EDTA - 4 mgm. BAL per kg. is given initially and followed in 4 hrs. by BAL in the same dose plus simultaneous EDTA 12½ mgm. per kg. These drugs can be given I.M. and should be given every 4 hours for a 4 to 5 day course. The patient is then given a rest for 3 to 7 days and the course of treatment may be repeated if specific tests remain positive. No more than 2 courses are to be given in one month.

Iron Poisoning - Immediate therapy should include induction of vomiting with syrup of ipecac followed by gastric lavage with 1% of sodium bicarbonate or milk. Desferal (Deferroxamine) 5 gms. may then be introduced for GI route by naso-gastric tube; and 92 mgm. per kg. is given intravenously SLOWLY in 100 cc. of 5% D & W over 1 to 2 hours. Rapid infusion produces hypotension. If the urine in the plastic collecting tube from the urinary catheter appears vin rose' colored, the patient has probably ingested a toxic dose and has over a 500 gamma % blood level. Such a patient may require 1 or 2 repeated intravenous doses at 12 hourly intervals.

Follow-up studies should include liver function tests; GI series for pyloric scarring; and eye examination for cataracts.

If Desferal is not available or the patient is in shock with massive GI bleeding, appropriate therapy would include whole blood transfusion; intravenous EDTA 37 mg. per kg. q12h; and exchange transfusion.

Lye and Caustic Poison - Esophagoscopy should be performed to see if the patient has esophageal burns. If these are present, he should be started on broad spectrum antibiotic therapy and prednisone in a dose of 40 to 60 mgm. per day. Treatment should be continued until subsequent esophagoscopy demonstrates healing of the burns. Generally, this requires high doses of adrenal steroids for a period of 1 to 3 weeks followed by tapering for a period of 1wk.

Follow-up examination includes a barium swallow at 4 to 6 weeks to detect esophageal stricture and/or pyloric stricture.

Organic Phosphate Poisoning - Parathion - Immediate treatment includes induction of vomiting (this is usually spontaneous); removal of soiled clothing and washing of the skin with soap to eliminate skin absorption of the material. Gastric lavage is performed with 5% sodium bicarbonate solution.

Immediate specific therapy consists of atropine 1 to 2 mgm. I.V. or I.M. and 0.5-1.0 mgm. every 20 to 30 minutes if the respiratory tract is not dry and there is lack of dry flush of skin, mydriasis, and tachycardia. PAM (Pyridine-2-aldoxime-methiodide) should be given in a dose of 50 mgm, per kg., up to a maximum of 2 mgm., as a 5% solution I.V. q 10 to 12 hours for 2 doses.

Barbiturate Intoxants - Ritalin in a dose of $\frac{1}{2}$ mg. per kg. I.V. slowly is the treatment for a patient in a coma from barbiturate intoxication. I.V. fluids and sodium bicarbonate therapy should be given.

Dehydration

Gastroenteritis -

General - Estimate the percent of dehydration by moistness of mucous membranes and by skin turgor. Estimate the electrolyte status by clinical appearance. Hypernatremic patients usually are extremely thirsty, have central nervous system irritability and their doughy skin turgor induces underestimation of the percent of dehydration. Hypo-electrolytemic patients are usually in a state of shock and have markedly diminished skin turgor. Isoelectrolyte dehydrated patients demonstrate reduced skin turgor; appear clinically alert, although on the verge of collapse; and are not excessively thirsty.

Fluids are to be given rapidly for the first hour, (400 ml/m²) and then slowed. Plan on replacement of the deficit being given over a period of 36 to 48 hours, with adjustments as indicated by the patient's clinical status. To determine fluid needs it is wise to devise one's own system and keep it understandably simple.

Specifics:

Water - less than one week of age - maintenance 75 cc. per kg.

Age 1 week to 1 year - 120 to 130 cc. per kg.

Age 1 to 2 years - 90 to 110 cc. per kg.

Age over 2 years - 75 to 90 cc. per kg.

For deficit, add 45 to 50 cc. per kg. for each of two 24 hour periods.

Sodium Chloride - maintenance is 2 to 3 meq. per kg. The deficit requires 6 to 10 meq. per kg., but it is to be given over a 48 hour period.

Please note that all figures are on the low side, which allows leeway for increasing the amounts as indicated by the patient's clinical condition. Regard normal saline as being a 1% salt solution with approximately 15 meq. per 100 cc. It is best to start all therapies with one-fifth to one-third normal saline and 5% dextrose and water.

Sodium bicarbonate - should be used only if the patient's serum bicarbonate level is less than 8 meq. per liter. Therapy is directed at raising this level by only 5 meq. per liter. A dose of 2 to 3½ meq. per kg. is usually sufficient to accomplish this (1 ml 7½% = .9 meq.)

Potassium - potassium chloride should not be started until the patient is voiding; and when used can only be given in a dose of 3 to 4 meq. per kg. with not more than 40 meq. per L. concentration. This dose equals maintenance and replacement.

In hypertonic dehydration, the patient needs mainly water; no hydrating load; less and slower fluids, only three-fourths the total volume; and less salt, one-fifth the total volume.

Diabetes Mellitus

The diagnosis of a diabetic condition - mellitus or insipidus - should be suspected by a history of preceding polydipsia and polyuria; enuresis resuming after bladder training; and nocturia. Any comatose, dehydrated patient who is urinating during physical examination has polyuric dehydration, and must be considered as a diabetic.

Specific Therapy - After the diagnosis is established by blood and urine analysis and the I.V. is started, the patient should be given regular insulin, 2 units per kg. if he is in a coma. Half of that is to be given intravenously and half subcutaneously. Another dose of 0.5 to 1.0 unit per kg. should be given after 1 to 3 hours. If the patient has mild ketoacidosis, a dose of 0.6 to 1 unit per kg. is sufficient.

Intravenous fluid should be given rapidly for a period of 2 hours and should consist primarily of saline, or saline and 5% D & W. This fluid is to be given at a rate of 2% of the patient's body weight per hour for 2 hours if he is in coma, and half that rate if he is not in coma. Sodium bicarbonate is also required at a dose of $3\frac{1}{2}$ meq. per kg. If 5% D & W is administered, the patient should be given regular insulin to cover the dextrose; 1 unit per 1 to 2 gm. (25 to 50 cc.) of dextrose, subcutaneously. The total fluid replacement should be 5 to 10% of body weight in 6 to 8 hours for the patient in coma, with half that value for the non-comatose patient.

After 2 to 3 hours, intravenous potassium should be added to the fluids, as described previously.

Subsequent doses of regular insulin can be determined by urine sugar or blood sugar determinations every 2 to 4 hours. If urine sugars are used, the patient should be given 3 to 5 units for every plus over a 1+ urine sugar value. After 3 to 4 days, the patient should be stabilized sufficiently so that the preceding day's insulin may be totaled up and he can be given 75% of that value as regular insulin one-third to NPH two-thirds; or all as NPH. Subsequent days' dosages will be decreased as the patient's muscle activity is increased.

TREATMENT OF BURNS

Alvin P. Mancusi-Ungaro, M.D.*

Burns are still one of the severest catastrophes that can happen to any individual. They probably require the most intense care and the most thorough back-up with hospital facilities of any major injury. Burns have been discussed over and over again, and everyone is complacent in thinking that he can handle them well. Yet, all one has to do is look at some of the results, including my own, that have been far from satisfactory.

What we are really trying to accomplish when we treat a burn? We are trying, with minimal risk in the most rapid and intense way, to restore the integrity to the skin and to preserve life, function, and appearance. How you do this will have to vary from individual burn to individual burn. We are familiar with the first-aid treatment and transportation and, of necessity, will limit our discussion in these fields.

Contamination

One of the greatest problems with burns is the contamination of a burn. The smearing of butter or ointment, as a first-aid measure, is going to alter the burn, making the diagnosis and further treatment more difficult. We teach, in the emergency treatment of burns out in the field, that the cleanest, occlusive dressing (sterile sheets) should be applied to protect the wound and, if possible, a splint utilized. Once the patient is admitted to the emergency room, you have the problem of treating him.

The More Severe Burn

Let's consider the more severe burns first. You have to preserve an airway, alleviate the pain, start anti-shock therapy, and try to get the patient out of the emergency room as soon as possible. Obviously, the emergency room is usually not a clean area. If a major burn is contaminated with pseudomonas bacteria, you may eventually have a mortality on your hands or, most surely, you will prolong the reconstructive phase. You must treat the shock immediately, and shock is frequently insidious in burns. The patient can look well but suddenly you will find that his blood pressure is falling, his pulse is rising, etc. Next, check with the first-aider who brought him in, to get a history. Because of the differences in treatment of electrical burns, steam burns, chemical burns, friction-type burns, and flame burns, ascertain how the patient was injured. Each type acts differently and must be evaluated differently. Take a history. Find out if the patient is a diabetic. Note any significant facts, such as, does he have a good heart, are his kidneys functioning well, etc. Next, one should estimate the extent of the burn. I think we all know

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the rule of nines in adults. Approximately 9% of the total body surface volume is occupied by the face, head and neck; 9% by the upper thorax and 9% by the lower abdomen anteriorly, that's 18%; 9% for the upper and 9% for the lower back, another 18%; 9% for each upper extremity and 18% for each lower extremity; and lastly, 1% for the perineum; a total of 100%. If the emergency room facilities are good, this is the time to determine the percentage and depth of the burn. If they aren't, the patient should be taken to a sterile room such as an operating room, have the contaminated clothes, etc. removed from the burns, and begin definitive treatment.

Major Differences

There are major differences between a first degree burn, which is just a simple erythema; a second degree burn which is vesiculation and extends deeper into the dermis; and a third degree burn, which is a total skin loss. Early, that is one or two hours after the burn, one of the best ways to differentiate between a second and a third degree burn is by sensation. If you stroke the patient with a sterile piece of gauze and he experiences pain demonstrating that the nerve endings still remain, he has a second degree burn. If the area is numb, it is probably a third degree burn. Once skin edema has set in, the second degree burn, which should have sensation, becomes numb also and one cannot differentiate by this method. If the burned area is absolutely blanched and white, it is probably a third degree burn. If it has a red hue, it is probably a second degree burn. Now, what is the clinical significance? A third degree burn should be re-surfaced as rapidly as possible. The body's protective envelope has been destroyed and bacteria can invade, leading to cellulitis, septicemia, kidney destruction, etc. If it is a second degree burn, it behooves you to use proper treatment, so as not to convert it to a third degree burn. If you wrap it in dressings and increase the temperature by preventing the normal evaporation mechanism from working, you can, in the presence of a small, low grade infection, enhance the infection and convert a second degree burn to a third degree burn. Thus, you are compounding the injury. You will probably increase the bacterial colony count unless your agent is bacteriocidal. This leads to increased toxicity.

Electrical Burns

Electrical burns are usually severe. Do not underestimate them. The prognosis should be guarded. There is a point of entrance and a point of exit. If it is in the hand, you will probably see a burned point where the current has entered, and if the patient is grounded, say at his foot, it will exit through this foot. Electrical burns, being very deceptive, may look amazingly mild when first observed. In a few days, when the burn starts demarcating, you will see the true picture. One of the more difficult electrical burns we see is in electric socket burn to the mouth. This occurs when a youngster takes the unplugged socket and puts it in his mouth while the other end is still in the outlet. Of course, he gets a sudden burn. This is not only destructive of soft tissue, but frequently burns the teeth, including the secondary tooth buds. For example, little may be seen immediately but, two to three days after the accident, the whole corner of the mouth may be sloughed out. The tongue may be burned and the secondary tooth buds may be destroyed.

In chemical burns with no prior treatment, obviously the best thing is to wash them copiously with water, rinsing the chemical off as quickly as possible. If you haven't time to use the proper neutralizing agent, such as an acid for a base, etc. please use plain water, irrigating over and over again.

Flame burns are usually dry burns. They can be much deeper than is obvious. Because of the flame, the high concentration of heat is at a localized area and the burns are usually deeper, involving the soft tissue beneath.

The steam, hot water type of burn, conducts heat to a larger area, and so usually involves a greater area in the injury. In these burns, you see a lot more vascular thromboses, and have varied problems with vascular changes. An ultraviolet burn, from the sun, or, a flash burn, from a stove, is usually quite superficial.

Prognosis

We have to evaluate the age, the general condition, and the total amount and type of burning of the patient. A twenty percent third degree burn in a 60 year old person is usually fatal. A younger person will probably recover without too many complications. If proper management is instituted immediately in any burn, up to about thirty percent of the body surface, in people under fifty years of age, the mortality rate is low. As you go above fifty years, and, as you go over a thirty percent burned area, the prognosis rapidly worsens. Infants cannot tolerate burning at all. Have a pediatric consultation as soon as possible. These children have little tolerance for the shock that ensues.

Site Important

The site of burning is important. There is a tremendous difference between a burn on the abdomen and a burn on the hand or face. Characteristically, a small burn on the back of the hand can produce a large amount of secondary contracture and loss of function. In the normal hand, we note that if we extend the fingers, we can easily pick up the apparent excessive skin on the back of the hand. If you hold this skin tightly and then try to make a fist, you will find that you can't because you are immobilizing the transverse palmar arch. In order to flex this arch and let the thumb opponate to the fifth finger, one has to increase the surface volume of the posterior aspect of the hand by 50% from the extended to the flexed position. This type of injury will limit the thumb motion of opposition to the index finger and possibly the middle finger, and you have virtually a crippled hand. Thus, when examining the burned hand, ask the patient to make a fist. You will now see the true extent of the burn defect.

The face acts similarly. Where we have free borders like an eyelid, nostril or mouth, a small amount of full thickness skin loss can produce dire contractures that are very difficult to repair once the deep fibrosis has set in. These must be repaired as soon as possible, and the only method is adequate resurfacing with skin. Obviously, we should choose the donor site skin with respect to color, texture and donor site scarring as well.

Encircling Burn

Another type of burn that one has to be extremely careful about is the encircling burn. Any burn that encircles an arm, or leg, or chest wall, will, if it is deep enough, start contracting and produce a tourniquet effect. These burns must be evaluated carefully and you may have to do escharotomies and, or, fasciotomies. If treatment is delayed, one may end up with an ischemic necrosis or Volkmann's contracture. If the forearm is completely encircled with a burn, watch for sensation in the fingers as well as circulation. With the onset of paresthesias, be suspicious that deep edema is occurring and, if the paresthesias progress, and, if scattered areas of numbness appear, the patient should be taken to the operating room and a releasing fasciotomy performed. In the defect that has been created, with the muscles bulging through the surgical incision, place a split thickness graft as a temporary physiologic bandage. If the wound were left open, the deeper structures of the forearm would likely become infected and, fibrosis and muscle contracture around the nerves would result with all the sequelae that go with this type of injury. Therefore early fasciotomy is used if it is necessary. This concept is similar to that of a tracheostomy. One either needs it or one doesn't. If you think you need it, do it.

The same holds true for the tracheostomy. When evaluating the patient, establish a good airway in the emergency room, give sedation if required, and start fluid replacement. Of course, tetanus antitoxin and supportive measures are also instituted. Probably the safest general all-around electrolyte is one-sixth molar sodium lactate. This is especially true if you have a patient with an incomplete medical history. Once the intravenous is running, place an indwelling catheter into the bladder so that the urine output can be monitored. Probably, the best guide of renal function is maintaining the urinary output at about 50 cc per hour. If you can maintain this for the first twenty-four hours, you're probably in an area of relative safety. With respect to tetanus, one must use big doses because of the magnitude of the injury. Be sure there are no allergies lurking. If there are, use hyper-tet or one of the medications less liable to produce an allergic reaction. This is precisely the time to evaluate the antibiotic therapy, if indicated. If there is a combination of burn-crush-avulsion injury, you probably should start an antibiotic of a broad-spectrum type.

Clinical Observation

Look at the patient. Is he obviously a negative protein balance individual, or, is he an alcoholic? Remember that the laboratory work obtained in an attempt to monitor the blood and chemicals only measures the substances in the circulating plasma at the particular instant the sample was drawn. It is not a true reflection of the total potassium, or the total sodium, or the total proteins, etc. Use your powers of clinical observation. If feasible, take the clothing off in the emergency room, as it shouldn't be dragged through the hospital. It is usually dirty and contaminated. This is also the time to take photographs to document the case, if a camera is available. All documentation should be accurate and your clinical impression of the patient is essential. A camera may be kept in the emergency room to be used for this purpose.

Airway Problem

If there is an airway problem, and, especially if the burns involve the neck, it is probably safer to have an endotracheal tube placed in position than to cut a tracheostomy incision through the burned area. However, if you are not capable of inserting an endotracheal tube and no one is present who can, then a tracheostomy must be performed, if indicated. Management of tracheal toilet is essential. We, as plastic surgeons, request that you learn to do a tracheostomy with a horizontal skin incision. These are much less likely to keloid or hypertrophy, for we all know that transverse incisions of the neck heal exquisitely well, whereas vertical ones will result in a thickened scar. These severely injured patients always require special duty nurses. Nursing care is absolutely essential.

Burn Care Center

As a nation, we are just beginning to develop the concept of burn care centers and as such, we will have specialized personnel in specialized physical plants. A true burn center will have a section where nothing else but burns are treated. It will be a sealed unit, either under positive or negative air pressure. This will tend to carry contaminants away from the patient. Everyone entering will change his clothes, will have an air wash on his body and will put on a sterile gown, gloves, etc. Remember, we are attempting to attain a good, sterile environment that is supportive in nature, because infection produces greater mortality in burns than anything else. If you can prevent this infection, a goodly portion of these burns will heal spontaneously. Do everything within your power to limit infection and contamination. This means sterile bed sheets, a single patient in a single room, isolated; it means good technique going in and out, with gloves, gowns, etc. A lamellar air wash bed is probably ideal, for, after several hours in this air wash, the patients total skin surface is sterile. Also the humidity is maintained properly.

Morphine

If much pain is present and morphine is required, please don't administer it intramuscularly. These patients are probably in shock or going into shock, although, it may not be clinically obvious. The morphine may then pool, and the patient will not be relieved by the drug. Later, perhaps another dose will be administered, and, when the patient suddenly comes out of shock, all the morphine is suddenly mobilized by the reconstituted circulation and severe cardiac respiratory depression may result. Thus, it should be given intravenously if indicated.

Physiological Approach

Most important at this stage is the psychological approach. Don't look at the patient and say, "Oh my God - what do we have here?" You are going to drive him into deeper shock by superimposing psychological shock upon clinical shock. You may establish a distorted body image and so disturb his psyche that the patient will be very difficult to handle. Utilize the Art of Medicine. Calm, positive reassurance is indicated. However, don't commit yourself by

stating, "You are going to be okay; you're going home in a couple of days". Instead you may state, "You are in a fine center, we're going to have a lot of experts in, we are all going to help you, you may have a difficult burn, sure, but don't worry about it. Everybody is going to do everything to help you, and I think things will work out well." State it positively so the patient believes it. In the dehumanizing practice of modern medicine, we tend to forget these things. If you implant fear in the patient and reinforce it, it is going to be very difficult to undo the severe depression you have created. You may destroy the patient's will to live; especially in a woman, if her face is involved. Therefore, use your best bedside manner.

Physiologically speaking, perform a cutdown and place an intracath in the vein. Try not to use a needle, as many fluids are going to be pushed into that vein, and you are going to develop a phlebitis, etc. Thus, while you have the chance, use a good catheter. Even though you started early with fluid therapy in the emergency room, the patient may enter shock by the time he gets up to his floor, the veins will collapse and you will have an even bigger problem on your hands. A general fluid formula that is safe to use, remembering that formulas are only as good as the judgment behind them, is the Brooke Army formula. This was developed at Brooke Army Center, in San Antonio, and is really quite simple. $1\frac{1}{2}$ cc of electrolyte-containing fluid and $1\frac{1}{2}$ cc of colloid, plasma or blood per kilo of body weight are each multiplied by the percentage of body surface burned times the weight in kilos. To this volume you add about 2000 cc of water because of the patient's insensible loss via perspiration, breathing, etc. This gives you the total fluid replacement for twenty four hours. However, the maximum of surface burn used in your calculations is 50%. Even if the person is 90% burned, you must never use more than 50%, otherwise you will overhydrate him. Once you have ascertained this figure, you must now adjust according to the following. If the majority of a 40% burn is second degree, you probably will not need quite as much; if it is all mostly first degree, go very cautiously so that you don't overload the vascular system of the patient. If it proves to be mostly third degree you have a much more severe problem present. The isotonic form of sodium chloride is widely used. This tends to increase the acidosis that occurs following burns. This is especially true in a diabetic. Therefore, we suggest one-sixth molar sodium lactate instead. For colloids, one can use plasma, whole blood, dextran or serum albumin. Whole blood should be used only to replace whole blood loss.

Remember, one of the mechanisms that occurs in a burn early is hemo-concentration. If you fill up the patient with red cells, when you really need proteins, you are going to create a severe problem. Thus, use only whole blood when you need whole blood. If you need proteins, utilize proteins not red cells. Usually one or two transfusions are helpful early in the severe burn. Take cultures immediately, realizing how long it takes, time-wise, to have a culture and sensitivity test returned. Thus, start with a broad spectrum antibiotic, if it is indicated. Look at the burn with a Wood's light. If the burned surface floresces, you have pseudomonas bacteria present, as they will show up in a Wood's light two or three days before they are clinically obvious; that is, before, you get green drainage of a purulent nature. If they are found to be present, you can start the proper antibiotic because, as is well known, a pseudomonas septicemia is almost 100% fatal. Today's usage of garamycin which is

specific for pseudomonas, has improved the prognosis substantially. If one gets a green urine (verdo globin uria) you must assume you have a full blown pseudomonas septicemia with probably many abscesses in the kidneys and, of course, the prognosis is most grave.

Open Dressing

I, myself, prefer open dressing every place except on the hand. Recently, I visited a burn center where they treat all patients at all times under open treatment. When a patient enters the unit, as standard technique, he is placed in a tub and washed with pHisoHex. I don't think this should be done for we know that if you have a deep second degree or third degree burn, and use pHisoHex to wash the patient, it is absorbed through the granulation tissues and it concentrates in the spinal fluid and can suddenly produce convulsions due to the meningeal irritation. A spinal tap would reveal pHisoHex concentrated in the spinal column. Thus, we do not use pHisoHex on third degree burns or deep second degree burns anymore. Plain soap and water is all that is needed to cleanse the wound properly. One can then decide whether the sulfamylon treatment or the silver nitrate treatment will be used, or a combination of both. In the sulfamylon treatment the cream should be gently spread on so that it forms a layer about an eighth of an inch thick over the entire burn and it should be changed at least daily. Recently, documented research showed that sulfamylon in animals inhibits epithelial regeneration in controlled burns. Thus, it is conceivable that if sulfamylon is used, a second degree burn may be converted to a third degree burn, or at least it's ability to regenerate epithelium would be markedly inhibited. Silver nitrate, one-half of 1% is an ideal solution, but it is messy, and it is hard to handle.

In the average burn plain saline is preferable. The burn has been washed down with soap and water, the patient is placed on a sterile bed in a sterile environment and gauze flats are laid over the burns directly and wet with saline. These gauze pads are changed, depending upon the problem, every two hours to every eight or ten hours. One must be cautious for, as the fluid evaporates, they can chill the patient. Warming blankets may have to be used. Each time the nurse, under sterile technique, takes these gauze dressings off, she is really doing a minimal debridement. Thus, she will keep the wound beautifully clean and the colony count of bacteria to a minimum. The patient is started on around-the-clock physiologic saline gauze dressings, one layer thick, and they are removed every two hours, day and night. Usually within 48 to 72 hours the wound is as clean as can be and is ready for grafting. Much nursing care is involved. Good technique is indicated to prevent further contamination. At this time pig skin, commercially available grafts, are laid on the third degree burns as an ideal physiologic dressing. These are removed every fourth or fifth day and a new crop can be put on until the patient is sufficiently stabilized so that autogenous skin grafting can be performed. This has the effect of cleaning the wound efficiently and producing an excellent base for the patient's own skin.

If homologous skin is used, it is preferable to use the mother's skin for the child, as in 25% these cases will show long term acceptance. Contractures of the hand are difficult to overcome later, and, as such, it behooves us to

put the hand in position of function immediately. Thus, the wrist is 25 degrees dorsi flexed at about 5 degrees ulnar deviated with all fingers flexed in all joints, including the knuckles, and the thumb in opposition and equidistant from each finger tip. Thus, the longitudinal and transverse arches are both preserved in this position. If the injured hand stiffens to a great degree, this is the ideal position of function for this type of injury. Therefore, I treat all burned hands with a closed method. It is well to remember one can't put a dressing on and come back ten days later and expect it to be healed. We all know that the sweat glands and sebaceous glands contain a normal bacterial flora. As one perspires a little under these dressings, the bacteria will grow up, out of the glands, onto the burned surface and produce a contaminant. If the occlusive dressing increases the temperature, one will breed infection and the exudate so formed can convert a second degree burn into a third degree burn. Thus, the dressings should be changed frequently. The remainder of the body is generally treated with an open technique. It is essential to be reminded that the perineum should never be treated with a closed bandage, as it tends to become infected.

Encircling burns of the extremities or chest are handled with a closed treatment because even though you are past the edema phase, secondarily the eschar coagulum contracts and exerts a tourniquet-like effect. Today it behooves us to replace skin loss with skin quickly. That does not mean three weeks later. In a third degree burn, the granulations are never sterile and never even clean. They are always contaminated. If one waits for secondary contractures to take place, it precludes good reconstructive surgery.

Technique Changed

The famous Copenhagen Burn Center in Denmark just released a superb paper. Prior to the early 1960's, they treated everybody coming into their institution, which services all of Denmark, with the closed treatment, silver nitrate, etc. They had a high mortality rate due to infections such as pyocyanosus streptococcus, etc. They changed their technique completely at this time so that every burn coming in is now treated with an open treatment. They are scrubbed down with soap and water, irrigated well, put on a sterile mattress and treated with a water soluble cream, if needed. Fluid and electrolyte balances are maintained. Each day the burns are debrided and by the tenth to fourteenth day they state that every burn is ready for grafting. By this tenth to fourteenth day, with total support for the patient, good medical balance, fluid balance, etc., the patient is taken to the operating room and enough skin is removed to completely graft the entire patient. These procedures may take as long as ten hours, and I quote them, "ten hours." In order to perform this surgery, the patient is maintained in excellent shape and a team is constantly monitoring him. Even a cardiologist is present in the operating room. They take as much skin as they will need for the total resurfacing of the patient at that one anesthetic. If the anesthesia proves to be too prolonged for the condition of the patient, they may awaken the patient in his bed, if need be, and lay the skin on that they have previously removed in the operating room with no suturing and no dressing. Every six hours somebody checks it and if there is a little seroma that develops, they lift it, nick it, and release the seroma. The skin graft begins to take within three days. They have found that their mortality rate has come down, but more important, their

morbidity has dropped. Their patients go home very early now and it is rare that they cannot completely graft a person in one operation. Their main residual problem is the encircling burn that involves the back and the front, for example. Obviously, they cannot graft both the back and the front at the same time because of the patient's positioning. Thus, they take all of the grafts that they need from the patient under one anesthetic, keeping the anesthetics to a minimum, wake up the patient, explain what they are going to do, and just lay the skin graft on as you would a bandage, and observe the patient. They continually do this until the patient is completely resurfaced, using only skin taken at the original procedure.

Full Thickness Skin Graft

It should be remembered that full thickness skin should ideally be replaced with full thickness skin, not split thickness skin. Obviously, to take a full thickness skin graft, you have to create another defect and the patients usually can't stand this. There are certain places, however, where, if it is at all feasible, you should use a full thickness graft, that is, the eyelids, around the mouth or any free border, or on the palm of the hand or the working surface of the fingers, etc. If, for any reason, full thickness skin grafts cannot be utilized then split grafts, as thick as can be expected to take under these circumstances should be applied. You may now ask what determines how thick a graft will survive? The thicker the graft, the more nutrition it needs to survive. Therefore, you must have a better base to receive the graft. If you are dealing with an avascular type of base you should put on a thin split graft as a temporary bandage. It is really a physiologic dressing and you have to replace it at a later date with a thicker graft, because the thinner the graft the less dermis in it, and the more contractility, dryness, wrinkling and hyperpigmentation occurs. All of these tend to limit function and produce a poor cosmetic result. Thus, if the condition cannot feasibly accept a split graft, of a thick nature, one must utilize one of a thin nature but only as a temporary measure. It should be replaced before contractures develop.

Meshing Technique

If there is a fairly questionable wound where you think there might be a moderate amount of infection still beneath, you should mesh the graft. The meshing technique will expand a 4 x 8 inch skin graft to at least twice its area. However, the spaces created by the expansion will heal with cicatrix, not dermis, and this should be remembered. It is a good technique because if there is any purulent exudate, it will seep through the holes in the mesh and will not spread under the graft, destroying it. This mesh graft may tend to contract somewhat, but it can rescue you from a predicament. It is a neat physiologic dressing, at least as a temporary measure, converting an open to a closed wound. Another question is what should be used as a donor site? If one needs only one or two drums of skin, it is absolutely wrong to take skin from the thigh, especially in a girl, because it is an easy, available place. Obviously, you will create scarring on the legs when it is not necessary. Why not utilize the abdomen or the lower back? At least clothing will cover this scar at a later date. If you have to take ten drums of skin you will have to

use the front, the back, and every place available. Electively, please try to choose the skin that is most likely to match in color and texture with the recipient site, utilizing a donor site that does the least damage.

Where to Treat

Simple or small burns can be treated in the emergency room. If it's only a superficial second degree burn, use an ointment as this will keep the air off and prevent pain. With a first degree burn, you don't really have to do anything. Ask the patient to protect it and stay away from heat and further exposure to the noxious agent. If it is a deeper burn of any significance, the patient should be admitted and all definitive treatment should take place in the operating room or in the hospital under sterile technique. If the patient is in such poor condition that you can only graft a certain portion, graft the most important areas first not what is most convenient and handiest under the surgical drape. The joint spaces, axillary webs, the neck, the face, ears and the hands require first grafting. With reference to ears, remember that herein we have skin almost directly on cartilage which is very susceptible to a chondritis. If the ear is extremely swollen, use extra caution for, if it swells sufficiently to compress the little arterioles, cartilage may be liquified within 24 hours. The entire ear will collapse. The ear must be drained to decompress the system thereby preventing an ischemic necrosis. Of course, don't let the patient sleep on the injured ear.

In conclusion, our treatment for burns today is not much different than it was thirty or forty years ago except that we do know more about the electrolyte balance and about the chemical physiology, we have better drugs to utilize, and we are more capable of taking care of the bacterial contamination. It is well to remember that we are always treating a total patient.

CARDIO-PULMONARY RESUSCITATION

Charles R. Ream, M.D., F.A.C.P.*

I am going to talk about cardiac arrest as it appears in a hospital setting, and I am going to divide this into four specific sections. The first will be the diagnosis, the second will be the emergency treatment of the cardiac arrest, the third will be definitive therapy, and the fourth will be post resuscitative care.

The first two of these, namely diagnosis and emergency treatment, are often already under way by the time the patient comes to the hospital. The family physician has often made the diagnosis and the emergency ambulance services have well trained personnel who are quite expert at doing emergency cardiopulmonary resuscitation as a first aid procedure. Patients arrive at the hospital with these measures being carried out. However, the physician at the hospital, in terms of refinements in diagnosis, emergency treatment, definitive therapy and of post resuscitative care, does the work that the physician at home and the ambulance squad personnel cannot do.

Diagnosis

Although the diagnosis has often been made before the patient reaches the hospital, I do want to touch on diagnosis and emergency treatment briefly and point up particularly those areas where we find errors have been made. First of all we will define cardiac arrest as sudden cessation of effective circulation. By this definition it does not necessarily mean that the heart has stopped beating. It does mean that there is no delivery of oxygen to vital organs and there is also inadequate uptake of the CO₂ that is being formed in these vital organs. The immediate goal is of course the diagnosis, and to attempt as quickly as possible to get cerebral reoxygenation on the way. One does not waste time trying to find an obscure etiology as I am sure you realize. One does not at this point take electrocardiograms. This is one great error that we find prevalent in the emergency room. The electrocardiogram will be used to diagnose an arrhythmia, but perfusion is all important and takes precedent. One should not at this point spend time with family and-or friend to try to determine what had happened. It is obvious that the patient has no pulse, that there are no carotid pulsations, that there is no discernable heart beat, and that when the patient is put in a prone position, he does not regain consciousness immediately. The breathing may be gasping, or there may be no breathing at all. The pupils are dilated, and we know that the patient is in the state that we call cardiac arrest. The diagnosis as to the syndrome is therefore immediately apparent. The cause at this point may or may not be apparent, but at this particular moment it is unimportant.

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Emergency Treatment

Now, let's talk a bit about the emergency treatment. Pupillary size is extremely important to us. Why? Well, it gives us some idea of how long the patient has been unconscious and perhaps how well we are going to be able to do in resuscitating him. It takes about 45 seconds after the heart stops and after cerebral circulation stops for the pupil to begin to dilate. It takes about 1 minute and 45 seconds from the time of cardiac cessation for the pupil to reach full dilatation. The brain is generally damaged if it is without oxygen from 4 to 6 minutes. If, when the patient arrives in the emergency room, the pupils are fully dilated, we know that we have already lost a minimum of half the time we have to reoxygenate the brain before permanent damage takes place. It is also important to note the size of the pupil because this is going to give us some idea as to how well our resuscitation is doing. If we are successful in what we are doing we will notice that the pupil will begin to reduce itself in size rather quickly. By the same token, if after one hour the pupils remain dilated, we have not gotten the pulse back, we have not gotten blood pressure back, and spontaneous breathing has stopped, then it means that our efforts should probably be discontinued. We spoke about the 4 to 6 minute time interval that we have to get this patient out of his dangerous situation. This is a relative amount of time because if the patient is anoxic before we started, or if the patient is elderly, or if the patient has cerebral arteriosclerosis or some other disease situation, it could very well be that our time instead of being 4 to 6 minutes, might really be only 1 or 2 minutes.

Call for Help

The first thing then that one does in the hospital setting is to call for help, because it is very difficult for one person to do resuscitation along. It is a tiring procedure and one needs help. You may need the anesthetists help, you need electrocardiograms to be used at a later time, and you need people to help relieve you in the resuscitation. You need oxygen therapists as well as nursing personnel. You need other physicians for cut-downs, etc. So we start off by calling for help. The first thing one does is give the precordium a sharp blow with the fist. (Demonstration about force used in blow to chest). If the patient is fortunate, sometimes that is all you need, and you can start up the heart again. Most of the time however, the patient is not this fortunate and you have to go further. You then begin artificial ventilation. Now I think the biggest mistake—the biggest error—is that we do not pay as strict attention to ventilation as we should. Cardiac Intensive Care Units are great, but because the word Cardiac precedes the words Intensive Care, there is a great tendency to only treat the heart, when in fact we are required to treat the whole person and all his physiological functions together, because each physiological function affects the other. We treat acid-base balance, the kidney, the lungs, the brain, and the heart, to mention only a few systems, and all at the same time.

In my experience the biggest error made in cardio-pulmonary resuscitation is failure to ventilate the patient properly. One fault is that we do not get adequate and proper airway, and we do not get an adequate airway because we do

not do what Dr. Abellana is going to show you now. (Demonstration-proper method to get airway and intubation. Errors in technique shown and consequences of same.) (Dr. Ream resumes lecture.) This must be done quickly and expertly. I want to qualify this, because it always depends upon the condition of the patient, really, as to what you are going to do at this point. One cannot carry out the exact same procedure on every patient. Indeed if one could, we would not need doctors. I am sure that you have seen patients in cardiac arrest that are gasping. The heart has stopped, but they have this: (Demonstration of sound). In these patients you do not have to worry about respiration first. In these patients you would go ahead with the cardiac massage first, but airway must follow immediately.

Cardiac massage must be done properly. (Demonstration of cardiac massage, ventilation with ambibag showing how personnel can get into trouble with airway while cardiac massage is going on. Demonstration on manikin and cutaway manikin). For effective ventilation the chest must move. (Demonstration). If one listens with the stethoscope, air can be heard moving in and out of the lungs. (Demonstration). Intubation is most desirable, and all emergency room physicians should be expert at this. However, we are often in a situation where it is most difficult to do from a technical point of view. If intubation cannot be accomplished immediately, one should not waste time. Emergency tracheostomy or tracheal needle may be necessary. One more word about intubation. If you are immediately successful, make sure you have the end of the tube positioned properly so that it aerates both lungs and not just the right side. Also, make certain that it remains in the proper position and does not move position because of cardiac massage. (Demonstration). Take your stethoscope and listen over the chest to see that you are getting air on both sides. Listen to the abdomen to make sure you have not intubated into the stomach. (Intubation demonstrated with pitfalls one can fall into without proper technique and equipment.)

A word about respirators. One cannot use respirators when doing cardiac massage. The pressure of the machine is not great enough to overcome the pressure that is being used on the chest when cardiac massage is being carried out. Most machines in use today do not deliver a fixed tidal volume that you can regulate. When cardiac massage has been stopped because adequate cardiac function has been restored and the patient is put on a respirator, you must be certain the tidal volume and rate is proper, and that acid-base balance is properly maintained. As shown in the demonstrations, respirations are performed at a rate of 15 per minute, and cardiac massage at a rate of 80 per minute. If you are all along, you usually give the patient three good breaths and then you go ahead and give him 15 good massages, then you go back and you do the same thing over again. (Demonstration of entire cardio-pulmonary resuscitation. Attention called to errors of most frequent occurrence by Dr. Ream as demonstration proceeded. The following were commented upon as the demonstrations were carried out:

Pupil response and clinical meaning.

Suction before airway inserted.

Adequate ventilation.

Technique of getting adequate airway.
Intubation technique.
Technique of cardiac massage.
Rate of massage and respiration.
Degree of pressure to chest. Amount it must move.
Cardiac contusion, liver contusion, liver rupture, spleen
rupture, fractures ribs discussed.
Infant cardio-pulmonary resuscitation discussed and demonstrated.
Efficiency of the procedure.
Dangers in pressing carotid areas when feeling for pulse.
Femorals preferred.

Definitive Treatment

We will now proceed to discuss definitive treatment. If there is cessation of heart beat, I think it is agreed by most that the empirical administration of .5 milligrams of epinephrine I.V. is the procedure of choice. It may be given intra-cardiac, and when this is done you usually use the fourth intercostal space, approximately 2" lateral to the sternum with a needle, #22 gauge, 3½" long. You inject into the ventricular cavity and not into the ventricular muscle mass, so that you must get blood back after the needle is inserted. Sometimes, just the stick of the needle alone will start a heart off. Sometimes, it will break a ventricular fibrillation and the heart will come back to a normal sinus rhythm. It is important, of course, at the same time to get cut downs going if one cannot get into a vein immediately. If one can get a vein, the faster this is done the better. At the same time that we do this, we begin to treat metabolic acidosis at once. The way we do this is to give sodium bicarbonate, and we usually give one 50 ml. ampule which is 44.6 milliequivalents. We give 50 ml. of that solution every 10 minutes I.V. Usually at this point, we have patients who are hypotensive. The drugs that we ordinarily use are Aramine, using 200 mg., in 500 ml. of 5% dextrose in water, and drip at a rate that is effective in getting blood pressures back to acceptable levels. Now you come to the point with some of these people where you have such severe hypotension that they are in profound cardiac collapse.

We see cardiac collapse often after myocardial infarction, and we see it after cardiac arrest. Effective therapy is really lacking in these situations, but we do the following: We start off with Aramine or Levophed and if they do not work we next go to very large doses of steroids and give solucortef in the amount of 3 grams in one bolus I.V. We have had some patients respond under these circumstances, but the number is few. There may be hypovolemia, and if this is the case, efforts should be directed at attempting to correct this undesirable state by giving expanders. Our present knowledge concerning therapy for profound cardiac shock is not solid. Cardiac assist may be indicated for this situation and as you know this type of therapy is in an experimental stage now.

Let us now turn our attention to the monitor. The EKG machine documents the arrhythmias we are dealing with although this can often be diagnosed clinically. Therapy is then used accordingly. If we are dealing with ventri-

cular fibrillation, then of course the method of choice in correcting this is electrical shock. The same therapy is generally in order for correcting any of the arrhythmias (not blocks) that we might be dealing with at the time of acute myocardial infarction, particularly in shock because we have got to correct them fast if we want to correct the shock.

First I will talk about ventricular fibrillation and the way that we would treat this. Then we will go to the monitor and heart sound machine and we will show you what various abnormal rhythms look and sound like. We cannot demonstrate the sound of ventricular fibrillation because you cannot hear anything, there is no blood pressure and the patient is always unconscious. The EKG looks like this. (Demonstration) Let us assume that our patient is in ventricular fibrillation. There are really two types; one is a fine fibrillation and the other one is coarse fibrillation. (Demonstrated). If we have fine fibrillation then we make an effort to convert it to coarse fibrillation. The way that this is done is to give Epinephrine as we stated before .5 mgs., or 5 ml. of a 1 to 10,000 solution. We then wait to see if we convert to coarse fibrillation. (Cardio-pulmonary resuscitation is going on at this time). If we do, then we defibrillate using a DC current of between 100 and 400 joules, usually about 100 to start with. If that does not work then we would up it to 200 joules. If that does not work, up to 300 joules. If that does not work, to 400 joules. (Demonstrated). If that does not work, then frequent shocks, placing one of the electrodes on the sternal area and the other one around the apex, or even further posteriorly. (Demonstrated). Let us assume however, we still have fine fibrillation, even after giving Epinephrine, 5cc of 1 to 10,000. Then we give calcium, and we give this in the amount of 5 ml. of a 10% solution of calcium chloride, and if we are fortunate we will convert to a rough or a coarse fibrillation; we will shock, but our chances for success are not good. So much for ventricular fibrillation.

Let us return to the patient who presents with myocardial infarction. Posterior wall myocardial infarctions often have rhythm problems that anterior wall infarctions do not have. Posterior infarctions tend to have bradycardias, and bradycardia may be a forerunner of premature ventricular contractions. The premature ventricular contractions that are spaced out at good intervals often become more frequent. Instead of coming from one focus they appear to be coming from two or more foci. We prefer now to say multifocal, instead of saying multifocal. At any rate we begin to see some of them that are up, and others that are down. (Demonstrated on monitor). The next thing that usually follows this is a run of ventricular tachycardia. (Demonstrated). This may go right into ventricular fibrillation. (Demonstrated). When these situations occur, and probably most of them occur outside the hospital, the patient usually dies. In bradycardias with myocardial infarction, atropine is indicated, isoproterenol may be used instead but may also cause ventricular arrhythmias. In the hospital we always worry when we get the slow rate because we have seen this pattern of bradycardia-ventricular arrhythmias result in death so often. I like to put in a demand transvenous pacemaker as soon as we can and then manipulate the pharmacological agents at our disposal. (Types of pacemakers and catheters demonstrated).

Now we would like to demonstrate a few of the arrhythmias to you on the scope and let you hear how they sound by auscultation. The first one that we will demonstrate is the premature ventricular contraction. (Demonstration). In patients that do not have heart disease premature contractions by themselves do not mean anything. They are of no particular clinical significance. In other words, if a patient comes to you with a PVC and thinks he has heart disease, then you have to find other evidence of heart disease before you say this is significant. We must have history of angina, history of dyspnea, and an EKG that is abnormal, something of this nature. In fact, probably most people after age 50 have them. Some of these people, oddly enough, do not feel them at all, and they can have a great many. Other people, if they only have one, are conscious of them. On the other hand if we are dealing with someone with arteriosclerotic heart disease or a myocardial infarction the PVC becomes of great significance to us because it means that we may be getting into a situation that is telling us that we are about to have ventricular tachycardia or ventricular fibrillation.

Ventricular tachycardia is usually defined as a rhythm where one has three or more of these complexes in a row. If that is the case then the patient should certainly be treated, and the drug of choice will be lidocaine, procaine amide, or quinidine SO_4 , depending on the clinical setting. There is some debate at this point as to whether or not a patient should be treated if he was having say, three PVC's a minute, and if he had had a myocardial infarction. Some physician's might wait before they treated at this point, until five or more a minute appeared. If block is present or if it is suspected it will occur, waiting is understandable, for the drugs mentioned in the presence of block are generally contradicted.

There are four types of tachycardias that one encounters, namely: Sinus tachycardia, atrial tachycardia, atrial flutter, and ventricular tachycardia. (All demonstrated on EKG and by sound with effect of carotid pressure. Clinical differentiation at the bedside without EKG discussed and demonstrated. Heart block demonstrated on EKG and auscultation. Adams-Stokes discussed.)

After resuscitation attempts have been successful we are usually faced with three major problems, namely: Acid-base balance, renal shutdown, and repeat arrhythmias. In the emphysematous we have all of these complicated by further respiratory problems. The acid-base problem if not corrected, inevitably leads to adverse arrhythmias. Proper ventilation frequently corrects this problem. Acute tubular necrosis not infrequently follows shock and can be missed if one does not look for it. Urine volume per hour is most important to know, and urinalysis before a catheter is inserted is of great help. If urine output is good, regardless of how low the blood pressure reads out, the patient is not in any amount of serious shock. Mannitol has a definite place in therapy, but it should be remembered that if the patient is in failure or close to it, the drug is contraindicated. The management of renal shutdown is a topic by itself and time does not permit us to make more than this brief mention of it.

The Future

We have done through a great many facets in the case of the patient that requires cardio-pulmonary resuscitation. Coronary care units have been set up in most hospitals to cope with these problems. I believe that the future will see the end of coronary care units per se. The patient is a whole patient with many organ systems all dependent on each other for total function. Intensive care units for the critically ill whether it be from heart attacks, medical hemorrhage, severe trauma, and-or surgical hemorrhage, burns, sepsis, respiratory decompensation, renal decompensation, etc., will be more and more the case with a solid corps of specialists in nursing, and specialists in the medical, surgical, pediatrics, obstetrics and gynecology disciplines working together in this area. Radiologists and pathologists will all be involved very actively with such units. The initial contact of the patient with the hospital and such units, is with you, the emergency room physician whose initial diagnostic acumen and professional knowhow will determine in no small detail whether or not the patient ever arrives in the unit I have just spoken of.

CARDIAC EMERGENCIES

Richard Daniels, M.D., F.A.C.P.*

This program is a training program for emergency room physicians. However, it is a course in the medical treatment of emergencies in general and is not limited to emergency room physicians.

My topic is cardiac emergencies, including pulmonary edema. I plan to cover the treatment of acute pulmonary edema, the treatment of cardiogenic shock, the treatment of cardiac arrhythmias in myocardial infarction and without myocardial infarction.

Pulmonary Edema

One must be able to recognize acute pulmonary edema in its various forms. In reviewing the patho-physiology of pulmonary edema, you may remember that the precipitating factor, usually is acute left ventricular heart failure. This can occur as a chronic or acute phenomena. With the advent of acute left ventricular heart failure, there is an elevation of the end diastolic pressure in the left ventricle, consequential elevation of the pressure in the pulmonary veins, elevated pressure in the pulmonary capillaries, and then an increase in the fluid in the interstitial spaces. The hydrostatic pressure exceeds the oncotic intracapillary pressure and forces interstitial fluid across the capillary membrane into the interstitial space. The first event that occurs clinically is the phase of interstitial edema which is manifest by a very mild amount of dyspnea. This may not be clinically significant and only may be ascertained by the use of X-ray. On X-ray, at this very early stage of acute pulmonary edema, one would see a butterfly pattern of perihilar infiltration.

Following this, if the left ventricular end diastolic pressure remains elevated, the next thing that occurs is alveolar edema. In other words, the fluid now seeps through the alveolar membrane and begins to fill the alveoli. At this point one would begin to notice mild cough, somewhat frothier, and the patient would have rales in his chest upon auscultation. Also, he will probably not be too comfortable.

As the patho-physiological process progresses, the stage of paroxysmal nocturnal dyspnea comes next. With this, the patient begins to develop bronchospasm. At this point he may sheeze audibly. A stethoscope on the chest may reveal diffuse wheezing, bilateral ronchi and rales at both bases. Chest X-ray would show an increase in the perihilar infiltrations and even perhaps diffuse infiltrations throughout both lung fields.

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Then, if untreated, the patient would go into the final stage to acute fulminating pulmonary edema with which you are all familiar. The patient is now coughing profusely, and is unable to lie flat because of dyspnea and orthopnea. He is tachypneic, the cough is productive of thick, pink-tinged mucoid serous type of fluid and there may be audible rattles on inspiration at the bedside. Auscultation of the chest at this point would reveal coarse rales on inspiration throughout both lung fields, as well as the ronchi on expiration.

The patient would appear in the emergency room or your office with this, and of course, the first thing to do is to try to oxygenate the patient. He may be somewhat cyanotic at this point. If tolerated, oxygen by mask is probably the treatment of choice. Very frequently, patients are extremely anxious and will not tolerate a facial mask and therefore a nasal catheter or a prong nasal cannula must be used. The patient must be sedated, and the drug of choice for sedation in this instance is morphine sulfate. It is best given in a I.V. dose of 10-15 mgm and may be repeated every hour or two until the patient is in a state of somnolence. The rationale for the use of morphine is twofold: one, it allays anxiety, which is very often an aggravating factor in acute pulmonary edema; and secondly, it interrupts the Hering-Brewer reflexes which produce more tachypnea. The presence of fluid in the alveoli irritate the lung and set off the Hering-Brewer reflexes, produce more tachypnea, producing more transudation of fluid. One might choose Dilaudid for sedation or analgesia because of the absence of side effects which sometimes accompany the use of morphine, i.e., sinus bradycardia and gastro-intestinal upset.

The effect of gravity should be utilized in order to keep the fluid in as dependent an area as possible. The patient should be treated in the sitting position, either sitting in bed, or sitting in a chair. If one can effectively trap the extracellular fluid in the vascular space, and reduce venous return to the heart, this will reduce the amount of load on the right and therefore on the left ventricle. This is the rationale for the use of the rotating tourniquets. The tourniquets should be applied to three extremities and rotated every 20-30 minutes. We now have, in most emergency rooms, the hydraulic rotating tourniquet machines which work very effectively. As an alternative to rotating tourniquets, phlebotomy may be done. The phlebotomy of 500 cc of whole blood is frequently a very effective adjunctive therapy.

The use of aminophyllin is important where there is a large bronchospastic element present. Therefore, where some wheezing or rhonchi are heard, predominantly, I.V. aminophyllin in a dose of .25 to .5 gm would be most effective.

In order to administer the I.V. medication that we have mentioned, and will mention, it is probably a good idea to start an I.V. at the outset of therapy. Open a vein, put up a solution of 5% dextrose and water and let it run very slowly, just to have a vein available for the administration of I.V. medication. The morphine maybe given through this, the aminophyllin may be given through this, and then the digitalis which we are now going to discuss, also may be administered in this manner.

Assuming for the moment that the patient is not, or has not been recently on digitalis, it is probably efficacious to digitalize rapidly the patient. Many patients with acute pulmonary edema will not require I.V. digitalis. However, if after the use of morphine, rotating tourniquets, oxygen and aminophyllin, the patient shows no response, then cedilanid or another rapid acting digitalis preparation should be given I.V. My personal preference is for the use of cedilanid and it should be given in the total dosage of 1.6 mg divided into either 2 or 3 doses. Generally speaking, it is wise to give half the digitalizing dose, which would be 0.8 mg. I.V. at once when the patient is first seen. After waiting 3 or 4 hours another 0.4 mg may be given, then after another 3 or 4 hours, another 0.4 mg may be given. As an alternative, other rapid-acting digitalis preparations would be Ouabain in a total dosage of 1 mg I.V., or digoxin in a total dosage of 1 mg I.V.

I.V. diuretics also are effective and they may be used initially in acute pulmonary edema. If the patient does not respond to the aforementioned modes of therapy, I would then go to I.V. ethacrynic acid. This also may be administered through the I.V. which we now have open and is given in the dosage of 50-100 mgm, depending upon the size of the patient, in 50 to 100 cc of dextrose and water. It is given in a period of about 15 to 20 minutes. The I.V. ethacrynic acid works very rapidly as opposed to mercurhydrin or thimerin which have been used in the past. The onset of action of ethacrynic acid is about 15 minutes and it will produce generally a 2500-3000 cc diuresis over a period of about 3 hours, maximum effectiveness being about 3 to 4 hours. With the use of ethacrynic acid you must be sure to watch potassium loss. It is a potent kaluretic agent, and if the patient is concomitantly being digitalized, one must be careful about getting into trouble with hypokalemia and cardiac arrhythmias. I.V. Furosemide, in a dosage of 40-80 mgm, may be equally effective.

A newer mode of therapy for acute pulmonary edema has been the introduction of glucogen. Glucogen has a potent positive inotropic effect on the heart, and in some circles it is thought to be almost as effective as digitalis. Glucogen increases cardiac output without increasing peripheral resistance as do some of the other catecholamines. It does not cause myocardial irritability as does digitalis. In the future it may be the drug of choice in acute pulmonary edema. Glucogen acts very rapidly when given I.V. It acts at about 3 minutes and is fully effective at 7 minutes; the duration of effect is about 15 minutes. It is rapidly picked up, rapidly mobilized by cardiac muscle and rapidly excreted. The dosage of glucogen which is being recommended is 2-3 mgm per minute given over a period of 5 minutes, for a total dosage of 10-15 mgm given I.V. over 5 minutes.

If the patient is severely hypertensive with acute pulmonary edema, one may use a ganglionic blocking agent. The drug of choice is Artonad 250 mg in 250 cc of 5% dextrose and water, given by intravenous infusion to lower the arterial blood pressure by 30%, titrating the rate of infusion against the blood pressure. The average is 25 ml. per minute (10 drops per minute).

In the treatment of acute pulmonary edema, one must look for a precipitating cause. Usually there is a precipitating cause, and after one has gotten through the emergency situation and has time to pause and reflect upon the cause, one must look for one of the following causes for precipitation of this acute cardiac decompensation. Causes are: infection, whether it be pulmonary infection or systemic infection, and consequent tachycardia; infarction, either pulmonary infarction or myocardial infarction. Arrhythmias are frequent causes of acute pulmonary edema, either supraventricular arrhythmias or paroxysmal ventricular arrhythmias. These, of course, must be treated concomitantly or else the pulmonary edema will not clear. Other precipitating causes of pulmonary edema are hypertensive crisis, acute elevation of BP, and fluid overload in a hospital situation where the patient is being treated with blood or I.V. fluids.

Cardiogenic Shock

Cardiogenic shock is found in approximately 10 to 20% of patients with myocardial infarction. There is pump failure present. That is, failure of the myocardium to produce adequate cardiac output, as opposed to electrical failure, the most common cause of death in acute myocardial infarction. Cardiogenic shock is not a single blood pressure level, but is a clinical syndrome and is manifest by the appearance of the skin, which is cold and clammy, and usually pale and cyanotic. The BP is usually low, but may be what we consider normal, if the patient had previously been hypertensive. The pulse is thready, there is tachycardia, and there is an altered state of consciousness. The patient may be lethargic, he may be confused, and he may be agitated. Urinary output is usually low or absent.

The hemodynamic events which occur in cardiogenic shock are responsible for the clinical syndrome. The myocardial infarction will produce acute myocardial damage in the left ventricle and this will reduce the stroke output of the left ventricle causing a decrease in cardiac output. The decrease in cardiac output causes decreased perfusion of the organs which are vital to life. That is, decreased perfusion of the brain will alter the state of consciousness; decreased perfusion of the kidneys will produce oliguria or anuria; and decreased perfusion of the coronary arteries will precipitate further myocardial damage and arrhythmias. In an attempt to compensate for decreased cardiac output, there is usually an increase in the heart rate, which is inadequate to raise the blood pressure.

The peripheral vascular resistance may be increased or normal during cardiogenic shock, and the central venous pressure may be normal or increased, depending upon the state of the left ventricular function. The central venous pressure is occasionally low in cardiogenic shock, and we will talk about that when we discuss volume replacement therapy. The arterial oxygen saturation is generally low, the PCO_2 is generally low because of hyperventilation, unless the patient has associated emphysema and the pH is usually low because of the effect of lactic acidosis. The total blood volume is normal or increased if congestive heart failure is present.

When one sees a patient in cardiogenic shock, one must consider and rule out other causes of shock, in order to be sure that the shock state is due to a decreased cardiac output or myocardial damage. In considering the differential

diagnosis of cardiogenic shock, one must be sure that the patient does not manifest one of these other causes of hypotension or shock. They are as follows: acute neurogenic shock which is neurogenic syncope, and may be due to acute pain or anxiety; cardiac arrhythmias associated with shock, that is, ventricular tachycardia; blood loss shock due to acute hypovolemia, not too uncommon in patients on anticoagulants who may have an acute episode of GI bleeding. A less common cause of shock in such patients would be acute adrenal insufficiency. That is adrenal hemorrhage in a patient who has been on anticoagulants. Pericardial tamponade caused by pericardial effusion or hemopericardium might produce a similar picture; and ventricular rupture also would produce this.

Pulmonary embolism might be associated with a shock-like syndrome, as well as rupture of the intraventricular septum, rupture of a papillary muscle or rupture of a chorda tendinae. These last of the aforementioned diagnoses which are potentially treatable causes of shock are treated in a different manner from the idiopathic cardiogenic shock associated with MI.

In the treatment of cardiogenic shock, one must remember that early treatment is effective treatment. The later the treatment, the less effective the treatment. Hemodynamic studies are important to differentiate the different patterns of cardiogenic shock. These are the measurement of central venous pressure, continuous measurement of cardiac output, if possible; and continuous measurement of arterial pressures. Careful surveillance of the patient's fluid balance, careful intake and output with measurement of hourly urinary output, with a Foley catheter in place is most important in determining the effectiveness of the therapy of cardiogenic shock. The outcome, of course, depends upon raising the BP and perfusing the kidneys and, therefore, getting an adequate urinary output. One knows that if the kidneys are excreting 1 cc/minute, that is 60 cc/hour, there is adequate perfusion and the cardiogenic shock is being treated effectively.

After the patient is placed in bed, lying flat or in a slight Trendelenburg position, a Foley catheter should be inserted and careful monitoring devices should be applied to the patient. A central venous pressure catheter should be inserted into the jugular or subclavian vein in all patients who are in cardiogenic shock. Serial measurements of the central venous pressure are important.

The best guide for total amount of fluid therapy are serial measurements of central venous pressure. These are easily obtained with a simple manometer and an intracath slipped in through the jugular or subclavian vein. Arterial blood gas studies for pH, the oxygen and PCO_2 should be drawn initially, then every half hour to every hour, depending on the patient's clinical status. The amount of alkalinizing solution given will depend upon the pH and PCO_2 .

Digitalis probably should be used in all patients in cardiogenic shock. There is a decreased myocardial contractility and a decreased cardiac output. Unless the patient had previously been on digitalis, or manifested digitalis toxicity, digitalis is one of the drugs of choice in the initial treatment of shock. Rapid incremental I.V. digitalization with Cedilanid in a total dosage of 1.6 mgm, as mentioned previously in acute pulmonary edema, or Ouabain in a

total dose of 1 mgm, should be given. However, the entire digitalization time should be much more rapid in cardiogenic shock because of the acuteness of the emergency. Initially, Cedilanid would be given .8 mgm, and perhaps 1 or 2 hours later another .4, then another .4, so that the total digitalization time might be 2 to 4 hours in cardiogenic shock. Glucogen may be very useful as adjunctive therapy in cardiogenic shock. The dosage would be the same as in pulmonary edema.

If the central venous pressure is low, a decrease in venous return to the heart may be an aggravating factor or a precipitating factor in cardiogenic shock. Therefore, blood volume must be elevated through fluid replacement. 10% glucose and water, or low molecular weight Dextran, should be given rapidly when the central venous pressure is low until the CVP rises to 10-12 mm. of water. At that point, 100 mgm of 10% dextrose and water would be infused rapidly over a period of about 15 minutes. If the central venous pressure rises and then falls to the base line levels of 10-12 mm. of water, one may assume that blood volume is low and continue to give increments of 100 cc of 10% dextrose and water rapidly over 15 minute intervals, until the central venous pressure no longer falls to its baseline level. At this point, one knows that the blood volume is adequate. This may be important in the patient who has been carried on a long term vasopressor or catecholamine therapy, which tends to deplete the fluid volume.

We will now discuss the catecholamines which are probably the most widely, but the least effectively, used therapy in the treatment of cardiogenic shock. First, we will discuss the various types of catecholamines that have primarily beta adrenergic effectiveness. The beta adrenergic effects are those that increase contractility and increase cardiac output, those that have positive inotropic effect on the heart. Other beta adrenergic effects on the heart are an increase in the sinus pacemaker, the production of a tachycardia, as well as peripheral vasodilatation. The alpha adrenergic effects of catecholamines are the opposite. That is, peripheral vasoconstriction with an increase in the cardiac work and perhaps a decrease in the cardiac output.

The major hemodynamic abnormality in cardiogenic shock is the failure of the heart as a pump. Therefore, it is reasonable to use the beta adrenergic drugs in such a situation. The primary beta adrenergic drugs are: isoproterenol (Isuprel) and mephenteramine (Wyamine). These drugs have the major effect of producing increased myocardial contractility and increased cardiac output. However, they may precipitate serious arrhythmias because of the increased myocardial irritability. They also have a peripheral vasodilator effect, as mentioned previously, which may lower arterial venous pressure. They are probably not the drug of choice. It is generally agreed that one should use a drug that has a beta effect on the heart and an alpha effect peripherally. That drug would probably be metaraminol which is Aramine. Aramine should be used initially in a dosage of 200 mg in 500 cc of 5% dextrose in water and should be given I.V. to titrate the systolic BP to 85-100 mm of mercury. If, after a reasonable time, 30 or 60 minutes, there is no appreciable effect on BP, Levonorepinephrin (Levophed) should be substituted. This is used in the dose of 1-4 ampules, which would be 2-8 mgm per 500 cc of dextrose and water. If

neither Aramine nor Levophed are effective in raising the BP, one might then try Isuprel, or Wyamine, and if one of these are not effective, then the use of epinephrine I.V. may be tried.

There are several newer methods of treatment. These methods, which are not applicable to all hospitals and situations, are hyperbaric oxygenation and mechanical devices that assist some or all of the pumping functions of the heart, including the intra-aortic balloon with counterpulsation, effectively used at hospitals where this gadget has been operating, and the use of artificial chambers with cardiopulmonary bypass or the artificial heart.

I might mention that massive doses of steroids are sometimes effective in the treatment of cardiogenic shock. Hydrocortisone, or its equivalent, may be used in the dosage of 30 mg/kilo; that is, a total of 1500 mg of hydrocortisone given rapidly I.V. or equivalent dosage of 500 mg of Solu-Medrol.

A drug that has been used experimentally in the treatment of cardiogenic shock, which is not available at this time, is dopamine.

Arrhythmias

We will now talk about the arrhythmias which may complicate acute myocardial infarction. In studies done with monitors applied to patients very early in the onset of myocardial infarction, it has been noted that approximately 90% of acute MI patients have cardiac arrhythmias. Many are undetected; however, this is the most common cause of death in acute MI.

When the patient appears in the emergency room with an acute MI, the immediate steps taken would be an I.V. to keep a vein open for the administration of drugs. Oxygen is administered and medication is given to relieve or allay anxiety and pain. Morphine Sulfate or Meperidine (Demerol) may be given. However, with the use of these analgesics, one must be careful to avoid hypotension and bradycardia. The effect of anxiety and of pain may precipitate cardiac arrhythmias, as may these drugs, as they precipitate bradyarrhythmias. One must watch for congestive heart failure, which may be accompanied by the complaint of tachypnea, dyspnea, and there will be a persistent sinus tachycardia.

In the treatment of the specific cardiac arrhythmias, one must consider whether or not the patient manifests evidence of congestive heart failure. There is an increase in mortality and an increase in the frequency of life-threatening arrhythmias where congestive heart failure is present. The lowest mortality and the lowest frequency of arrhythmias are, of course, found in the patient who does not manifest congestive heart failure. The highest mortality is in patients who are in severe acute pulmonary edema, and manifest cardiogenic shock. Approximately 95% of the patients who manifest cardiogenic shock have serious cardiac arrhythmias. The serious cardiac arrhythmias are as follows: either sinus bradycardia, or sinus arrest; second or third degree atrio-ventricular heart block; ventricular heart block; ventricular fibrillation; and asystole. The most frequent complications found in myocardial infarctions are the ventricular arrhythmias and conduction defects.

The best way to cope with a disaster is to avoid it. Doctor Bernard Lown has stated that each successful ventricular defibrillation represents a therapeutic failure in the prevention of ventricular fibrillation. We know that pain, anxiety and stress are potent initiators of dysrhythmias. We previously mentioned the use of morphine and Demerol, as being very effective in reducing pain. In addition, they also are effective in reducing the incidence of cardiac arrhythmias. Some feel that it is best to keep patients with acute myocardial infarction in a state of rousable slumber for the first 1 or 2 days in order to reduce arrhythmogenic anxiety causing release of catecholamines. Similarly, some use prophylactic anti-arrhythmic drugs routinely, although this is not generally held to.

The avoidance of valsalva maneuvers, the straining at bowel movement, are also valuable in prevention of cardiac arrhythmias.

In the management of cardiac arrhythmias in myocardial infarction, it is important to treat the patient and not treat the arrhythmia. By that I mean, if the patient has atrial fibrillation with a slow ventricular response, it may not be necessary to treat the arrhythmia. In a patient who has ventricular tachycardia associated with shock, if one treated the cardiogenic shock, the ventricular tachycardia may spontaneously remit. Therefore, one cannot state that there is a specific treatment for each specific cardiac arrhythmia. In general, I will try to outline the therapy of choice for each situation.

In discussing ectopic ventricular arrhythmias, we will first consider premature ventricular contractions. These do not necessarily have to be treated, but there are essentially four situations in which they should be treated in an effort to prevent more serious ventricular tachyarrhythmias. These are: (1) when the ventricular prematures exceed 5 per minute; (2) when there are multifocal ventricular premature beats; (3) when there are rallies of consecutive ventricular premature beats, 2 or 3 in succession; or (4) when there are early ventricular premature beats where the R waves in the premature ventricular beat fall on the downstroke of the T of the preceding complex. These four situations are harbingers of much more serious cardiac problems. Therefore, they should be treated immediately in an effort to prevent the serious ventricular arrhythmias. They should be treated with lidocaine (Xylocaine). This is the drug of choice and should be given in a dose of 25-30 mgm of 2% lidocaine as a bolus and should be followed by the continuous I.V. drip of Lidocaine in a dosage of 1-4 mgm per minute. If this is ineffective in abolishing the ventricular premature beats, then diphenhydantoin (Dilantin) may be used in a dosage of 50-100 mgm I.V. This may be repeated every 5 to 10 minutes to a maximum dosage of 400 mgm Dilantin until the prematurity is abolished. If both of these are ineffective, one may use Procaine amide (Pronestyl), giving 50-100 mgm increments every 1 to 2 minutes over a period of 10 to 15 minutes to a maximum of 1.0 gm of Pronestyl. With the use of Pronestyl, one must watch for signs of hypotension. If these are all ineffective, one may try quinidine. Bretylium Tolsylate, a new and not yet available drug, may become the treatment of choice in these situations. Bretylium in a dosage of 7 to 10 mg/kg (600-900 mg) is a potent antiarrhythmic agent with a positive inotropic effect at this dosage.

Ventricular tachycardia is the next most serious and most common cardiac arrhythmia. It occurs in about 20% of the patients with acute myocardial infarction and is manifest by 3 or more ventricular premature beats in succession. The ventricular rate is usually 120-210 beats/min. The diagnosis may be confirmed by the appearance of ventricular fusion beats or capture beats on the EKG. Where ventricular tachycardia appears in short bursts, lidocaine or Bretylium should be used in the previously mentioned dosages to prevent persistent ventricular tachycardia. If the tachycardia is persistent, and is unresponsive to Lidocaine, D.C. countershock should be used. Countershock should be used in a dosage starting with small energy bursts beginning at 50 watt seconds, increasing by 100 watt seconds to a maximum of 400 watt seconds. This is the treatment of choice in converting persistent ventricular tachycardia. If counter shock is used, 10 mg. of I.V. valium anesthesia may be used without an anesthesiologist. If countershock or lidocaine are ineffective, Propranolol (Inderal) may be used intravenously. Inderal may be used in a dose of 1 mg to 3 mgm. I.V. given over 15-20 minutes. When ventricular tachycardia is associated with cardiogenic shock or acidosis, one must correct these factors. Therapy often will not be effective unless the acidosis is corrected. If ventricular tachycardia appears in a patient who is on digitalis, and digitalis toxicity is suspect as the cause of the tachycardia, I.V. potassium may be used in a dosage of 80-120 mg in 500 cc of 5% dextrose and water, running in over a period of one hour. If this is ineffective, one may try EDTA, Dilantin or Propranolol.

Another method of treatment for ventricular tachycardia which has been effective is the use of electrical pacing. One may over-drive the ventricular pacemaker that is producing the ventricular tachycardia and capture the ventricular pacemaker and abolish the ventricular tachycardia. Accelerated idioventricular rhythm may occur during the course of myocardial infarction. This is a slow ventricular tachycardia, also called non-paroxysmal ventricular tachycardia. This probably does not require treatment. The patient usually, clinically, has no detrimental effects from accelerated idioventricular rhythms. When ventricular tachycardia occurs in the presence of complete A-V heart block, lidocaine is probably contraindicated. At this point the complete A-V heart block should be treated with Isuprel and use of a temporary transvenous pacemaker.

While the patient is being prepared for the passage of a pacemaker, if he had a long bout of ventricular tachycardia, lidocaine may be used to control these. However, very often the ventricular tachycardia is interspersed with periods of complete heart block, and in this case Isuprel may be used to relieve the patient of the heart block and therefore prevent tachycardia. Ventricular fibrillation is the final event which may result from ventricular tachycardia. Ventricular fibrillation is the arrhythmia which you should recognize on the EKG. The ventricles are usually beating irregularly; the pulse will not be palpable.

When a patient arrives in the emergency room without pulse or blood pressure and the ambulance personnel state that he looked fine a moment ago, we must assume the patient has had such onset of ventricular fibrillation. This will be right in approximately 90% of the cases. After a blow or two with the fist to the patient's chest, he should be given immediate direct current counter

shock without wasting time trying to obtain an electrocardiogram. If he is asystolic, you will not alter his chances by first attempting defibrillation. On the EKG, the ventricular fibrillation is immediate defibrillation: that is, countershock with a high energy, usually 200 watt seconds, and if not immediately effective, 400 watt seconds. The patient must be adequately oxygenated, with the passage of an endotracheal tube. The sooner that defibrillation is applied, the better the chances of cardiac resuscitation. The patient must be protected from anoxia. If the patient has had a cardiac arrest and is anoxic, he must be treated with cardiopulmonary resuscitation; he must be ventilated and cardiac massage must be instituted immediately.

Very often defibrillation will not be achieved de novo, and one must prime the heart with the intracardiac use of 5-10 cc of 1/1000 dilution of epinephrine. If not effective, the use of calcium gluconate may be used in a dosage of 10 cc of 10% solution directly into the cardiac ventricle. Again, I will remind you that acidosis must be treated with sodium bicarbonate given in a dosage of 500 cc of 5% sodium bicarbonate rapidly over a short period of time.

The atrial arrhythmias are not as life-threatening as the ventricular arrhythmias. Sinus tachycardia is present in about 50% of the patients with acute myocardial infarction and should be treated by treating the underlying causes of sinus tachycardia. That is, allaying anxiety, and treating hypotension or cardiogenic shock. Sinus bradycardia, is a sinus rate of under 60 beats per minute and is very common in diaphragmatic or posterior wall myocardial infarction. These are usually, but not necessarily, treated but should be treated if symptomatic or if the patient is manifesting cerebral symptoms.

Sinus bradycardia may be treated with the use of I.V. atropine in dosage of 1-2 mg given every 3-4 hours or with the use of I.V. Isuprel. Atrial or nodal premature beats are not necessarily treated unless they are over 5 per minute. These would be treated with quinidine or Propranolol, if treatment is indicated.

The paroxysmal supraventricular tachycardias that is, paroxysmal atrial tachycardia, and paroxysmal nodal tachycardia are often due to digitalis toxicity, particularly when they appear with some degree of heart block. Where digitalis is suspect, one must stop digitalis and treat the patient with I.V. potassium. If the patient is not on digitalis, manifests a rapid ventricular rate, and shows either coronary insufficiency on the EKG, or manifests congestive heart failure as a result of the rapid ventricular rate, then the paroxysmal atrial tachycardia should be treated immediately. The use of carotid sinus pressure may be tried first, but may not be effective. The drug of choice here is digitalis--rapid I.V. digitalization. Either cedilanid, Ouabain, or digoxin may be the drug of choice. If these are not effective, or if for some reason one may not want to use digitalis, Vaso-pressors may be used. Neo-Synephrine, 0.3 cc of 1% solution, diluted to 1.0 cc with non-saline, given intravenously to accurately raise the blood pressure will often break atrial tachycardia. Aramine in a dosage of 200 mgs. added to 5% dextrose and water given by rapid I.V. drip to produce sudden elevation of the blood pressure may achieve the same result. The alternate use of quinidine or cardio-version might be effective. If the patient has been on digitalis, one must be careful with the

use of cardio-version. The electrical energy produced with cardio-version is synergistic with digitalis and may produce digitalis toxicity and irreversible ventricular tachyarrhythmias.

Atrial flutter may be treated with cardio-version. This is probably the treatment of choice for paroxysmal atrial flutter. Atrial flutter, of all arrhythmias, responds best to cardio-version. Low energy currents are usually very effective. 10-25 watt second may be effective. If cardio-version is not selected or not effective, then the use of digitalis would be the next treatment of choice.

Atrial fibrillation is recognized easily on the EKG by fibrillatory waves usually appearing in a rate of 350-600 beats per minute. The ventricular rate is irregular. The treatment of choice where the ventricular rate is rapid is digitalization; again with the use of I.V. digitalis preparations previously mentioned. Direct current cardio-version may be tried in acute atrial fibrillation and is effective in about 30-50% of the cases. However, the patient must be maintained on an anti-arrhythmia agent, that is, quinidine or pronestyl, because 25-30% of the patients tend to relapse back to atrial fibrillation.

I will mention briefly conduction disturbances as the other complications that arise in the course of acute M.I. They are sino-atrial block, and various degrees of atrial-ventricular heart block. Sino-atrial block is manifest on the EKG by pauses in the P waves equal to 2 or more P-P intervals. It occurs in about 2% of the patients with acute myocardial infarction and is most often associated with inferior wall M.I. The prognosis is guarded because sino-atrial block and sino-atrial arrest are frequently confused with blocked atrial premature beats and sinus arrhythmia which do not require treatment. The problem with the sino-atrial block is that it may lead to sino-atrial arrest and cardiac arrest. Therefore, it should be treated with atropine in a dosage of 1 mg I.V. or isuprel in a dosage of 2 mg/500 cc of 5% dextrose in water given by I.V. drip until the atrial pacemaker picks up to a normal rate. Sino-atrial block may go on to a disastrous result with asystolic cardiac arrest and a pacemaker should be considered if unresponsive to atropine or Isuprel.

Some AV blocks do not necessitate treatment. The first degree of AV block, that is the PR interval which is over 0.2 second, probably does not require treatment. The second degree AV block may appear in two forms. Type I - second degree AV block is the Wenckebach phenomenon where there is progressive lengthening of the PR interval and shortening of the R.R. intervals until one beat is dropped. This may occur in a 1:2, 2:3, or a 4:5 ratio. This block is usually seen with inferior wall infarctions. These are often due to digitalis.

Type II, second degree AV block is described as the Mobitz type second degree AV block and is usually seen in anterior wall infarctions. This is where the PR intervals are constant but one or more ventricular beat is dropped. Third degree heart block is the appearance of complete AV dissociation with the appearance of a nodal focus (junctional) with a narrow QRS complex or a ventricular focus with a widened QRS complex as the ventricular pacemaker. Wenckebach AV blocks, if due to digitalis, may be treated with the use of potassium and the withholding of digitalis. The prognosis is good in these cases and a pacemaker may not be required. Mobitz type blocks are best treated with atropine or

isuprel and usually require a pacemaker particularly where the QRS is widened. While preparing the patient for a pacemaker in either high second degree AV heart blocks or 3rd degree heart blocks one should use isuprel in a dosage of 2 mg in 500 cc of 5% dextrose in water to titrate the ventricular rate so that the patient has no evidence of cerebral anoxia. One must be careful to watch for the evidence of ventricular tachycardia during the use of isuprel. Lidocaine may be kept handy to counteract such an occurrence.

Three methods are available for emergency pacing: (1) the Killip wire or transvenous pacing using flurosopic control; (2) a transthoracic pacing wire inserted through a needle into the left ventricle between the fourth and fifth intercostal space in the midclavacular line; and (3) a pericardial wire which is inserted between the inferior surface of the heart and the diaphragmatic pericardium. In the emergency room the transthoracic wire is probably the easiest method of pacing.

A question and answer period followed.

PSYCHIATRIC EMERGENCIES

J. Chernus, M.D.*

Definition

For you, who man the emergency rooms of general hospitals, the definition of psychiatric emergency must be pragmatic. A psychiatric emergency constitutes anyone who comes to your facility begging for immediate relief from distress which is obviously of psychiatric nature or anyone who is brought to your door by family, neighbor, employer or police, stating that the patient looked or acted, in a bizarre, or dangerous, or ominous fashion. Let me enumerate and describe briefly the most common situations you are likely to encounter and offer a word or two on what to do for them.

Attempted Suicide

First, the attempted suicide. This emergency runs the gamut, from an adolescent boy or girl who has swallowed a number of aspirins, to a middle-aged person, in a deep coma from barbiturates, tranquilizers, etc. The self-inflicted wound-type of attempted suicide runs the gamut from a superficial scratch of wrist, or neck to deep stab wounds of **chest**, or serious gunshot wounds or head, chest, or abdomen. Sometimes there is a two-pronged effort combining the swallowing of something poisonous as well as some form of self-wounding. Since the latter, because of the attendant bleeding, is the more obvious and dramatic, the doctor may neglect the oral episode. It is important from the medico-legal as well as from the purely medical point of view, to avoid this omission. To protect yourself, if the patient appears drowsy, ask him or those who brought him if he swallowed anything. Enter this on your record, as well as the fact that you examined the pupils, mouth, skin, etc. for evidence of drug ingestion.

I'd like to say, parenthetically, at this point that for some peculiar reason the person who brings the psychiatric emergency in have a pronounced tendency to slip away. They uniformly seem to be guided by the principle - "I got him here, Doc. - now he's your baby. So long." Be sure the nurse gets the names and phone numbers of these people before they get away. You may want to ask them about the circumstances in which they found the patient, even the kind of pills they took or the prescription (information) from the bottle.

As to management of the attempted suicide, the guiding principle is medicine first, psychiatry second. I am amazed how often the non-psychiatric emergency room physician gets "hung up" in his treatment of the patient because of the psychiatric angle. I think the reason for this is that the physician has not been sufficiently indoctrinated as to these priorities. He is perfectly competent to suture wounds, arrest bleeding, wash out the stomach, etc., but

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thinks that he must simultaneously ascertain why the patient wanted to kill himself and alter this state of mind favorably, right then and there. He often feels he can not deal with the problem unless the psychiatrist is summoned to be by his side at that very moment. He is especially likely to get "hung up" this way if the patient is still able to speak and floods the doctor with an agonizing tale of woe, usually involving the bad treatment he has received from spouse, family, or lover.

The doctor must be firm and clear. First, render the immediate physical treatment indicated - gastric lavage, antidotes, stimulents, dialysis, surgical treatment of wounds. There is time enough for the psychiatric angle later.

However, two questions do exist beyond physical treatment. One, should the case be reported to the police? Your decision should depend on the customary practice in your community and the seriousness of the attempt.

The second question - if the patient is in reasonably good condition after emergency treatment, should he be allowed to go home? Medico-legally, if the patient says bluntly that he wants to be admitted, he should be admitted for at least 24 hours observation, or should be sent to another hospital for admission on an emergency one-signature form. If, however, the patient insists on leaving, use this rule of thumb: Where there is a competent family group to clearly assume responsibility for the patient, let him go, with proper notation on the chart including the advice to consult with a psychiatrist. If the patient is alone or the person with him is not closely related or is indifferent, urge the patient to stay overnight. If he insists on leaving, make him sign an AMA release. Further medico-legal procedure calls for notifying the police that you have treated a would-be suicide who refused to stay in the hospital. If you decide on this, notify the patient that you are going to notify the authorities, as required by law. This may change the patient's mind.

Panic-Stricken Patients

The next most common emergency is a crisis situation (as perceived by the patient), which is not grossly psychotic. There are often the common-garden anxiety attacks - sensations of acute panic, nameless fear or the fear that "I'm losing my mind", or "I'm-like-fading away from myself." Bear in mind, parenthetically, that most of these cases, because of the subjective physical sensations, have turned up at your facility as presumed "heart attacks", "choking" or "impending stroke." With a little experience, you will soon get the knack of recognizing the "functional" nature of these "physical" emergencies. What I have reference to here is the "psychic" crisis. As I said, the prominent symptoms are extreme panic, fear of going insane, fear of losing control, sometimes concretized to fear "I'll do some harm to myself, my wife, or children."

The key to management is to render psychopharmacological and psychotherapeutic help. For the first, a therapeutic dose of a tranquilizer (Librium 50 mg, Valium 10 mg) by mouth or hypo will usually suffice. For the second, calm listening, not reassurance is the sine qua non. Don't say "you'll be all right," because in truth you really don't know. However, if you sit down with the

patient in a quiet room and calmly ask him to tell you what troubles him, assuring him by your manner that you are going to give him all the time he needs, it will work wonders because calmness is contagious just like fear. Even if the patient presses, don't give assurance or advice.

"Do you think it's serious, doctor? Can I lose my mind?" should be countered with - "I don't think that's important. The important thing is for you to get help."

Generally, you should talk with the patient at least a half hour. By that time usually he is calmed down and it is quite safe to encourage him to go home. Give him a single dose of a hypnotic to take and the names of several psychiatrists. Impress on the patient that he must call one of them to arrange for an early appointment. It is hardly ever necessary, in fact, it is unwise, to attempt to arrange a psychiatric consultation when the patient is in the emergency room.

Acute Psychosis

The next most common psychiatric emergency is the acute psychosis. You should be familiar with the two main types, schizophrenic and manic. Usually the schizophrenic is behaving peculiarly laughing, giggling, making bizarre gestures, or assuming strange poses, is hearing voices or expressing delusional ideas of grandiose, persecutory, or bizarre nature. The manic is in a state of high euphoria, talking a mile a minute. Each little item makes sense but the patient's thoughts flit from one subject to another. Sometimes the schizophrenic and manic attack are almost impossible to differentiate on first examination. This isn't too important in the emergency room. What is far more important is to differentiate the patient who is likely to break into assaultive or destructive behavior, from the one who is psychotic, but "harmless." This is difficult, but becomes easier with experience. The key is not which patient do I have to be afraid of, but which patient is afraid of me. The fearful patient becomes destructive and assaultive because he feels trapped--"cornered like a rat."

The most useful guide is the patient's expression and manner. If his eyes are shifting around as if to note the nearest exit, or to find something to grab to defend himself, or if he gives only partial answers to your questions, suspect him of potential "acting out." The distinction is useful in the management of the acute psychotic. If there is little danger of assault, flight, or destruction, sit down with the patient as described in the last section, but do not probe or discuss his psychotic material (delusions, hallucinations, paranoid suspicions, strange ideas, etc.) Nor should you probe into "dynamics," such as sexual experience, love relationships, philosophy of life, or childhood events. Instead talk to the psychotic patient as if he were normal. Ask him about his job, his vocation, baseball, television, etc. This line of conversation may demonstrate considerable healthy "islands" of functioning. If so, there is no urgency to get the patient admitted right then and there. You can prescribe a phenothiazine and send him home with his family with instructions that he be seen the next day by a psychiatrist. If there is a psychiatric unit in your hospital and the family is anxious to have him admitted, of course you admit him, as a voluntary patient.

With the other kind of psychotic, the one who is likely to erupt, you must sedate as soon as possible. Begin the task immediately - don't waste time in talking, or probing. Tell the patient that you can see he is ill and that it's your job to start treating him right away. "I'm going to give you an injection to calm your nerves. I'm the head doctor here, and it's my job to help you." Tell him this resolutely and firmly. In response to a prearranged signal your staff, including security guards, should approach the patient together. Give him at least 100 mg. of sparine or thorazine and arrange for immediate admission to the psychiatric section, or to another psychiatric facility, on a "one-doctor" emergency form. If the patient seems even more on the brink of erupting use $7\frac{1}{2}$ - 15 grs. of sodium amytal I.M. to be followed in an hour by the phenothiozine.

There are some experiments going on now with the use of mace to control patients who become violent. It will be useful to have a quick-acting, reliable agent to quiet an "acting-out" patient.

The true manic patient is never dangerous in this sense. The danger is in exhausting himself, sometimes fatally. Despite the advent of Lithium in the treatment of mania, the emergency room physician should not give Lithium. Better to quiet the patient with parenteral barbiturate, and/or phenothiozines. The manic patient should be hospitalized right away.

Agitated Depression

Occasionally an agitated depression is brought to the emergency room. He or she is usually an older person whose hyperactivity consists principally of pacing up and down and hand-wringing. Coupled with these actions, fears of poverty, being without basic needs or of having sinned, or "ruined" things, serves to differentiate them from the other psychotic states. Anti-depressants are too slow for emergency room management -- the treatment should be a therapeutic dose of a tranquilizer and a hypnotic. The patient can be sent home, providing there is a caring family, to be seen by a psychiatrist in the immediate future.

You should be on the alert for a spurious kind of psychiatric "emergency" - the elderly patient. Families sometimes bring an oldster to the emergency room, especially at night or on weekends. They will say he has suddenly become "senile" and can't be managed at home. The patient may be senile enough, but you can bet it wasn't of sudden onset. Often, the family has gotten wind that their oldster will not be admitted to the hospital through regular channels, so they try an "end run" by bringing him in as an emergency. Tactful discussion with the family, plus an offer to fill out forms for the State or county mental hospital, will usually resolve the problem.

The Alcoholic

The next emergency problem concerns the alcoholic. If he is merely acutely intoxicated and he has a good family situation, you can send him home to sleep

it off, after making sure there are no injuries present. If he is without suitable home and supervision, sedate him carefully with chloral hydrate and let him remain in the emergency room an hour or two to sober up. The alcoholic you must be careful about is the impending "D.T." case. He may come in, by himself, complaining of "the jitters" and ask for "just something to quiet me down." He may resemble the panic patient I described earlier, but experience will alert you to suspect he's an alcoholic. Ask him directly. He may be evasive, especially if he has no alcohol on his breath. But with the slightest history of alcoholism, assume that he is a candidate for delirium within 24 hours. Sedate him at once with 150 mg. of librium (for a man of average height and weight) and admit him without further ado. If he refuses, you must get an A.M.A. release and phone his family or friend.

Drug Addiction

The next item is the drug addict. If he comes in, or is brought in, near coma from an overdose of a drug taken in the pursuit of his addiction, the same advice is offered as for the attempted suicide -- vigorous medical treatment with psychiatric considerations subordinated until later. The trickier problem is with the addict who comes in with feigned complaints, begging for a shot of pain killer. Ask him what usually helps his pain. If you don't give your suspicion away, he will tell you - "demerol, or something like that." If there are no confirmatory signs of acute abdomen, you can be sure he's trying to "con" you into giving him a "fix". The variant of this problem is the admitted addict, who enters in withdrawal agony, begging for a fix. Tell him that you can give him an injection of a tranquilizer, but that if he wants an opiate or methodone, you must report it to the police.

Children

Lastly, you may have to deal with psychiatric emergencies in children, sometimes as young as age 12. The adolescent suicide attempt I have already covered. But you may also have to deal with youngsters who are brought because of a destructive rampage, uncontrollable rebellion or who have run away from home. The best service you can render is to talk alone with the youngster for 15 minutes to a half hour. Get his side of the story and make him realize he has a friend and champion in you. Then talk with the parents and the child, together. Point out to the parents that they have somehow lost their parental know-how; otherwise they wouldn't feel so helpless in dealing with their child. When both are quieted down, (give them a simple sedative) and send them home with the advice to get in touch with one of the staff psychiatrists or the Social Services Department. Occasionally, the problem is an epileptic furor. There is usually a history of prior seizures. In this case the child should be admitted for treatment of the underlying epilepsy.

TREATMENT OF MULTI-SYSTEM INJURY

William A. Dwyer, Jr. M.D. *

Multiple system injury in a single patient is one of the most challenging of diagnostic and therapeutic problems in the entire field of medicine.

I can still vividly recall my own introduction to this problem as an intern at Bellevue Hospital. On the first day I reported for work in my very clean and very white suit, and was assigned to the Surgery Section of the Psychiatric Ward. No sooner did I introduce myself to the Head Nurse, than a call came from the Emergency Room for the Surgical Intern. An elderly lady had jumped out of a fourth floor window and landed on the sidewalk. I arrived to find her body lying on the stretcher with obvious fracture deformities of one arm, both legs, the entire left rib cage was flailed. She had no blood pressure, was comatose, there was ecchymoses in both flanks, and she had vomited sometime during her transport to the hospital.

I can't begin to describe the feeling of absolute ignorance and helplessness that began to assail me, nor the wonderful feeling of relief when she took one last gasp and expired thirty seconds later. The many problems she posed were beyond any poor help I might have been able to offer.

The purpose of this lecture delivered some years and many patients later is to try to provide you, as Emergency Room Physicians, with at least a partial antidote to this feeling of helplessness, and to offer some guides to help combat the feeling of diagnostic and therapeutic ignorance.

I will limit my remarks to those measures necessary to the emergency care of the patient. As you might guess, this is a very vast subject and somewhere we've got to draw some limits. I make the presumption that other hands will take over to provide a continuation of care beyond the Emergency Room itself.

A Definition

I think we ought to define what we mean by multiple system injuries. This is a patient, who as a result of traumatic injury, has more than one organ or system damaged. This might be a burn with a fracture. This might be an abdominal wound with a G.U. complication, such as a ruptured kidney or ruptured bladder. It can be a head injury, plus a perforation of the abdomen. It can be a head injury with a fractured leg. There are any number of combinations. The point is that more than one organ-system is involved in the traumatic process. I also think it is important right at the outset to point out that the skin is an organ and a very vital one. Burns are an affectation of the organ-system of the skin. Now, it is quite possible, obviously, to have a patient with multiple lacerations of the skin and a fracture and the patient not be a big management problem. It would require some sutures; it would

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require setting of the fracture and that is the end of the problem. However, because of the two systems involved in the compounding of effects on the blood volume, etc., there are more problems than would be present with a simple fracture alone.

We will now discuss patients who are seriously injured, and will give you two examples:

First example: A 50-year old man was struck by a car while walking across the street. He had a fracture-dislocation of the pelvis with the pubic bone rotated 90 degrees out of position, a fracture of the tibia and fibula, comatose, no blood pressure, a laceration of his urethra, and a ruptured bladder.

Second example: A woman was admitted conscious but the ambulance attendant stated that she was periodically lapsing into unconsciousness. She had been involved in an automobile accident and had gone through the windshield, incurring a laceration of the neck. This laceration extended across the neck into the floor of her mouth, tearing the root of the tongue loose. The posterior pharynx was visible through her wound, she had multiple fractures of her ribs and a fractured femur. She had considerable blood loss with a blood pressure of (80) systolic. This is the type of patient we are interested in discussing.

Time is Critical

The problem in treatment of multiple system injuries is the brief period of time that one has to work prior to the patient's possible demise from any one of several causes. This requires an alteration in approach. The classic approach that we are all familiar with is a history, do a physical examination, determine what laboratory tests are needed to confirm our diagnosis, and then treat accordingly. With multiple injuries, time is of the essence and requires us to modify this approach. What we do first is a very complete but very rapid physical examination making sure the patient is completely naked. We examine him with specific things in mind. These specifics include the presence or absence of airway obstruction and the presence or absence of shock. By shock (what I mean and will constantly refer to as shock) is a lack of adequate circulating blood volume with impending irreversible circulatory failure. We must determine the presence or absence of major hemorrhage internally or externally which is causing shock and the presence or absence of fracture deformities or other organ-system disruptions that need imperative correction on an emergency basis. These are the things to which we must direct our attention immediately.

Treatment Priorities

This requires the setting up of a set of treatment priorities and requires us to examine the patient with those treatment priorities in mind. This is a little bit out of what you are usually accustomed to. You are examining to establish a treatment priority too. The first we've mentioned is the patient's airway. A patient can live for only three minutes with complete occlusion of the airway. He can live for a somewhat longer time with a partial occlusion. In the latter case, there is a problem of causing severe cerebral anoxia and brain damage, permanent in nature. Therefore, in every patient, search for

signs of cyanosis in the nailbeds, in colored patients particularly you must look at the nailbeds, look at the mucus membranes, look at the lips themselves, they have a characteristic appearance when compared to your own. Cyanosis produces a kind of violaceous hue instead of a bright pink. In respiratory distress this examination must be made in the very first few seconds that you see the patient. Additionally, the mouth must be opened and, if necessary, searched manually for foreign objects (chewing gum, dentures, etc.). It is amazing what collects in the mouth. This is especially true in all unconscious patients. We had a small child brought in unconscious not too long ago with a piece of chewing gum lodged in the back of his throat. The chewing gum lay there for a good three minutes before somebody had the presence of mind to put his finger in the mouth and get it out. Then the child woke up immediately.

Patent Airway

In any patient with a facial injury or badly broken jaw, there is a continuing threat of airway obstruction from hemorrhage flowing into the posterior pharynx; or from the tongue, detached from its usual moorings, falling back into the posterior pharynx. The unconscious patient also presents a problem in airway management because his normal reflexes are obtunded and his muscle tone gone. As a result, his tongue may fall back and block his airway, or he may vomit and aspirate his own vomitus. Complete airway obstruction prolonged beyond three minutes is fatal and partial obstruction prolonged for any length of time can result in permanent brain damage. Therefore, assurance of the existence of a patent airway is the first and most important task in any seriously injured patient.

Adequate suction machines must be immediately at hand to deal with secretions. In all patients where there is any question of continuing threat to the airway, this threat must be removed either by insertion of an endotracheal tube, or a tracheotomy; preferably, the former. Advocacy of an endotracheal tube is something of a departure from the traditional teaching of performing an emergency tracheostomy. It does have several advantages over tracheotomy, however. The morbidity of emergency tracheotomy under less than ideal conditions is considerable, and it is avoidable when you use an endotracheal tube. With a minimum of training, almost any one, including paramedical personnel, can be trained to insert it, and in faster time than it takes to perform a tracheostomy. Furthermore, this method avoids the direct introduction of infection into the respiratory tract.

The best method of establishing an airway is inserting an endotracheal tube; and the decision must be made quickly in the case of an unconscious patient. Be sure it has a cuff and is inflated. If there is any problem about respiration, hook him to a Bennett machine or respirator. On the Bennett machine, the patient initiates the breath, whereas on variations of the Bird respirator, the respirator does the breathing. There are two great dangers in putting patients on endotracheal -- cardiac tamponade and tension pneumothorax. You can put the tube in, but do not use positive pressure oxygen until you determine whether this patient has cardiac tamponade or tension pneumothorax.

Control Bleeding

Having ensured the patency of the airway and adequacy of respirators, attention must next be directed to control of obvious bleeding from whatever source, and preparations made to relieve hypotension. The use of pressure dressings for the immediate control of bleeding is obvious to all, as well as the emergency use of tourniquets for the control of severed major arteries. In passing, mention might be made of the advisability of using Potts' clamps or bulldog clips on severed arteries. These instruments will arrest bleeding from the artery without damaging the wall as does the regular clamp. This aids in the eventual repair of the artery in the operating room, because it helps to preserve arterial length. Of greatest importance, is the early installation of one or more large bore catheters for the administration of intravenous solutions and whole blood to restore blood volume.

Hypovolemic Shock

In any patient who has severe injury to more than one organ-system, the existence of hypovolemic shock must be presumed and measures taken to reverse it regardless of the recorded blood pressure. I could not emphasize any point more strongly than this. So frequently, I have heard, "when the patient came in she had a good blood pressure," so they didn't put an I.V.-in. You don't know what is going to happen in the next two minutes and blood pressure can disappear rapidly. While the patient's blood pressure is good, the veins are full and stand out clearly, you can get a catheter in with great ease. If you don't need a catheter, it can always be taken out; but, if you have to find a vein in a hurry, it can be an awful tough job, as you are well aware.

There are a number of ways that hypovolemic shock can present, depending upon the amount of blood loss, the general condition of the patient prior to trauma, his age, his physiological response to trauma, his other medical conditions, etc. Then, it is necessary to know and be able to recognize them all. The first type: limbs are cold, lips are pale, blood pressure is low, and pulse is slow. This is usually the type response you see to pain. Second type: lips are pale, limbs are cold, blood pressure is normal, but the pulse is rapid. Third type: lips are pale, cyanotic, limbs are cold, blood pressure is low, pulse is rapid and barely perceptible. Fourth type: lips are pale or cyanotic, the limbs are ice-cold, blood pressure is unobtainable and the pulse cannot be felt. Fifth type: lips are of normal color, the limbs are cold, the blood pressure is elevated and the pulse may be normal or rapid. Patients in this fifth group have a profound peripheral vaso-constriction in response to injury. This elevates the central systolic pressure which is what you measure, the pressure in the major arteries, but all of a sudden just as if you pull a plug, that blood pressure caves in. It will be seen that blood pressure is not a reliable guide to the existence of impending or actual shock. One little clue is provided in the feeling of the limbs. As shock progresses, limbs get colder due to vaso-constriction. If you feel limbs that are ice-cold, presume very quickly that that man is going to get into very bad trouble from hypovolemic shock pretty quickly. The best course of action is to presume extensive blood loss, install one or possibly two large bore catheters in categories 3, 4, and 5. A blood sample should be drawn at the same time - if necessary, by femoral vein

puncture, and sent for cross-match. Frequently, you can get a large catheter in, but you can't get a blood sample out because of the vaso-constriction of the vein itself. Don't waste time trying. Put an 18-gauge needle into the femoral vein, draw blood from it, and get your blood sample right away. You can waste a lot of time looking for another vein from which to get another blood sample. This is quick, and it is easy, with this method, I have seen no complications in my experience, and I would recommend it as an immediate source of blood sampling. Be careful you do not get the artery, please. By placing a finger on the arterial pulse and inserting the needle next to it medially, I cannot see how you can possibly injure the artery. Just stay medial to the pulse.

The laboratory routinely needs 45 minutes to one hour to properly cross-match blood. In the most desperate of circumstances, it will still require at least 15 minutes to get blood. This is cutting out the Coombs Test, etc., to give you blood in a hurry. Some replacement solution must be used in this interim. At present, the vast majority of surgeons favor the use of salt-containing solutions, either normal saline or Ringer's Lactate; not glucose in saline, but normal saline. These solutions will temporarily fill the defect in circulating volume without the risks (hepatitis and allergic reactions) associated with plasma. Dextran, both high and low molecular weight, has been used as a blood volume expander. However, there are many pharmacological reasons for not using either of these agents. The most important: one has an anticoagulant effect and the other, of course, causes an increase in blood viscosity. Both, of course, are undesirable in the shock state.

One additional word on drug usage in the traumatized patient. Neither vaso-constrictors nor vaso-dilators should be used under any circumstances. The routine administration of nasal oxygen or oxygen via endotracheal tube is desirable, as is the administration of sodium bicarbonate. Also essential in managing the hemodynamic problem is the insertion of a catheter for measuring central venous pressure. This can be threaded up the cephalic vein or inserted into the subclavia vein. It is then threaded into the superior vena cava. This is an invaluable aid, particularly in the elderly patient, in avoiding excesses in replacing blood volume.

Relief of Pain and Splinting

Having assured an airway and started fluids to replace lost blood volume, the next priority is the splinting of all fractures and the relief of pain. Splinting alone will help to restore blood pressure in many patients. Any sedating drug that you choose to use as demerol, morphine, etc. should be administered only by the intravenous route. Shock patients will not pick up medications injected into the intramuscular or subcutaneous area. They have a terrific vaso-constriction, there is no blood circulating in those areas and medication will not be picked up. Later on in the course of events, the patient is still in pain and somebody gives him another dose. The patient's hypovolemia has been corrected, he is now in a state of normal constriction and all of a sudden, both doses are circulating. This can cause profound respiratory depression. Last, I might caution that in the course of splinting, a check should be made for the presence of peripheral pulses; first, in the

major arteries, and as shock improves, in the more distal pulses. The absence of a peripheral pulse in one or another extremity, particularly when associated with fracture, is a grave indication for urgency in further treatment.

Last, measures to combat infection, must be initiated. Tetanus toxoid and/or human antitoxin should be administered and antibiotics started. This is the priority order and the various acts that must be carried out for any patient with severe, multiple system injury. These measures must be carried out rapidly and efficiently and in the order described previously regardless of whatever else may be wrong.

Problems in Specific Systems

Let us now turn to the specific systems and the problems they present initially. Head injury is far and away the most worrisome type of problem confronting the average physician. Apart from the mild concussion, the head-injury patient is usually semi-comatose or unconscious and provides little in the way of diagnostic clues as to the nature and severity of his cranial problem. Conversely, however, such an injury also requires very little in the way of immediate therapy unless there is some compelling neurosurgical crisis for which there are definite, easily recognizable physical signs. I might add as a corollary, hypovolemic shock or shock of any kind is never associated with a cerebral injury. This is not the cause of any shock, unless it is a terminal event in these cases. The most important sign to look for is a dilated fixed pupil on one side. This denotes a rapidly expanding blood clot within the cranial cavity which must be evacuated fairly promptly.

Next, the existence of motion of the extremities on one side, and not the other, implies a lesion or clot on the opposite side of the cranial cavity which may require surgical intervention. Third, the sudden onset of a semi-comatose or unconscious condition in a previously conscious patient, unassociated with any sudden drop in blood pressure or airway obstruction. This may well represent the onset of an epidural hematoma which is very very compelling, and as a matter of fact, one of the more dramatic types of emergencies that exist in medicine. This is an expanding clot, usually due to rupture of the middle meningeal artery. You have approximately three hours before you lose the patient, but it can be reversed by the proper neurosurgical intervention. The history you usually get is that the patient had a brief transient period of unconsciousness, woke up, felt fine, probably walked around the accident scene, absolutely had to be argued into the hospital, gets into the hospital and all of a sudden he is not making real good sense, and suddenly becomes totally comatose. Absence of motion in the two lower extremities must make one consider the possibility of transection of the spinal cord due to a back fracture or dislocation. Last, the presence of spinal fluid draining from the nose or ears, automatically implies a fracture through the floor of the skull or the temporal bone which may or may not require surgical intervention. Palpation of the skull with a sterile glove through lacerations, may well provide the knowledge of an underlying depressed skull fracture; also an indication for surgical intervention. Beyond these conditions, airway restoration and maintenance with a Bennett respirator coupled with care of the associated injuries, is all that is necessary for the moment. Antibiotics are, of course, indicated in every skull fracture.

Check the Chest

Lesions of the chest wall, and/or the lungs are sometimes referred to as "hidden" lesions, are very frequently missed in the multiple-system injury-type of patient (59 percent in one series that I reviewed recently). The other more obvious injuries distract attention from the chest so that a delay in prompt treatment results. Consequently, in the course of restoring the airway, the chest should be checked as well. Remove immediately any clothing on a patient, because you can't see anything and you might miss something important because it is hidden by a piece of clothing. This can range from something as trivial as a puncture wound with a piece of metal in it to a large laceration across the abdomen or a flail chest. So, all clothing should be removed as soon as the patient is admitted. Look at both sides of the chest for possible evidence of flailing of the chest wall. This is a visible inward movement when the patient inspires. As I inspire, my chest cavity rises. A chest instead of coming up normally, goes in, in response to the negative pressure being established in the chest cavity. That is a flailed chest. The side goes up, the flail segment goes in. The two lungs should be percussed to determine the presence or absence of pneumothorax - hypo-resonant sound indicates its presence. Oscultation is also of help. Absence of breath sounds or bronchial breathing speaks strongly for its presence. Where there is a question about it, an 18-gauge needle can be inserted through the chest wall and the pleural cavity aspirated. Put a little drop of saline on the hub of the needle and drop it into the chest wall. If the drop blows off, there is pressure in the pneumothorax; if the bubble is sucked in, this is a reasonably normal situation.

The presence of pneumothorax, of course, is an indication for the immediate installation of a one-way flutter valve or an underwater-seal type of drainage; the former is easier and less cumbersome to the patient. The presence of blood in the chest also can be determined by a physical sign and by needle aspiration. It should be remembered that air travels up, fluid travels down. The patient flat on the stretcher is not going to have blood anteriorly at the level of the second rib, he is going to have blood posteriorly at the level of the eighth or ninth ribs, and he is going to have air anteriorly. So if you are tapping a patient for air and blood, tap up here and back here. (Demonstration). Chest tubes also must be positioned posteriorly to drain fluid, and anteriorly to drain air. Don't expect that a chest tube placed anteriorly is going to drain the fluid out of the back of the patient's chest. To drain blood, the chest tube has to be in the back, to drain air up here. (Demonstration). The lung itself can be traumatized, resulting in a considerable amount of intraparenchymal hemorrhage, a so-called traumatic wet lung. For this condition, as for the flail chest, the most immediate need is endotracheal intubation and positive pressure breathing with a high oxygen content. I might add that the idea of external fixation, of flail chests with towel clips, etc., has been pretty well discarded. We now have available what is called a volume respirator, and running this at 60 centimeters of water provides enough oxygen and provides enough stabilization of lung, not chest wall, but lung, that we can overcome the physiologic affects of the flailing of the mediastinum which was so harmful in these patients prior to this apparatus.

Injuries to the heart and great vessels must be suspected in any case of severe chest injury.

Of particular importance, is the onset of cardiac tamponade with its accompanying signs of low arterial pressure, increasing pulse, weakness of pulse, low pulse pressure, and hypotension peripherally with rising venous pressure. You will remember that one of the things we discussed doing initially was the insertion of a central venous pressure monitor. If cardiac tamponade seems to be suspect, a needle can be inserted in the pericardial cavity, and aspiration carried out to confirm the presence of blood in the pericardial cavity. The best approach is the subxyphoid approach, the third into the pericardial sac. Attach one lead of an EKG to your needle. If you stick your needle into the heart muscle, you start to get an electric potential on your EKG and get a picture of myocardial injury.

Rupture of the aorta will result in massive bleeding and prompt death or the aorta will wall itself off enough to allow further diagnostic efforts, provided prompt hemodynamic support is initiated.

Abdominal Injury

Abdominal injuries always must be suspected in every multiple system injury. The prime examples are a ruptured spleen or ruptured viscus. Any obvious opening in the belly wall should be covered with towels soaked with saline, Bowel protruding through such a wound should not be replaced.

The classic findings of peritoneal irritation with tenderness, muscle guarding and rebound, show a need for surgical exploration of the belly. All these patients should have a N-G tube installed. In fact, a naso-gastric tube should go into every seriously injured person, whether he has abdominal injury or not. The patient is likely going to be on oxygen or forced positive pressure breathing and this is going to inflate his stomach, thus hindering his heart and breathing actions. The tube avoids the problem of acute gastric dilatation.

In the unconscious patient, the classic abdominal physical findings may be masked; however, a needle aspiration of the belly in the four quadrants may well reveal the presence of free blood, again an indication for prompt surgical intervention.

The existence of ecchymosis in the flanks speaks for a profound retro-peritoneal bleed. This may be from a torn aorta or other major vessel. Absence of one or both femoral pulses, even in the shock state, may be a valuable clue as to which artery is damaged.

Intraperitoneal injuries to the bladder are usually characterized by diffuse guarding tenderness and rigidity of the abdomen. Extraperitoneal ruptures do not have generalized abdominal signs; there is usually local swelling, pain and tenderness over the pubic bone.

A rectal examination may well uncover some bleeding in the rectum due to laceration of the sigmoid or rectum.

Foley Catheter

In all multiple system injuries, a Foley catheter should be installed in the bladder. First, this provides a way of measuring the patient's urinary output which is an excellent guide to the efficacy of therapy. Second, it provides a tent for any tears of the urethra which may be present. Third, it provides a urine sample which will give some clues as to damage of the G.U. system. The presence of an amount of blood in the samples obtained, gives some idea of the severity of damage to the kidney. Last, it provides a way of carrying out a cystogram which will in turn, provide information as to the presence or absence of a rupture of the bladder. A simple, although not too reliable test can be done in the emergency room. Put a measured amount of saline into the bladder through a catheter, clamping the tube for a few minutes. If you put in 200 cc and you get back 50 cc when you open the catheter, obviously it went somewhere - probably into the peritoneum or perivesicle space. Filming the bladder, or an IVP cannot reassure you that there is no rupture. You must put in some contrast fluid and take a cystogram.

Mention has been made briefly of the presence or absence of motion of the extremities. However, insofar as possible, in all patients without obvious fractures, this must be determined. Even in the unconscious patient, most of them will respond to painful stimuli by withdrawal. Pinching or sticking patients with a pin will cause them to move an arm or a leg, if it is possible for them to move it.

Lack of Motion

Lack of motion speaks for something seriously wrong, either a local fracture or a fracture of the spine, spinal cord compression high up or low down. A broken back is not obvious and until you know better, every unconscious patient should be treated as if he has a spinal injury. If such a fracture is present, special precautions must be taken with the patient to avoid adding further damage to that already present. The patient must be placed on a rigid surface. You may have to sandbag the head to prevent further extension of a fractured neck. You must take special precautions when moving this patient. Flexion of the affected area must be avoided, particularly with cervical fractures.

Splints should be applied to all local fractures as they are noted, even tying the arm to the trunk, or the good leg to the bad leg, will help temporarily. Fixation of the hand in position of function over a gauze roll on an armboard, will suffice temporarily for any **hand** fracture. Run your hands down the chest, quickly compressing the chest. This will tell you whether ribs are broken, and how many. Run your hands over the belly and press on the symphysis. Even a semi-conscious patient will let you know by groaning if he has a broken pubis. A pelvic fracture causes the greatest amount of bleeding and is the most dangerous.

Where bone protrudes through the skin, no effort should be made to replace it. This is a job for the operating room. These fractures should be immobilized as they appear; despite the deformity, don't try to replace it in position of normalcy. Splint it as it is, and try to immobilize it as it is.

Always check all the distal pulses; the absence of arterial supply, as I have mentioned, is a cause of increased concern and an indication for greater urgency of treatment.

Burns

Burns, as they come in, should be treated as hypovolemic shock. Have attention paid to airway maintenance with an endotracheal tube. There is nothing specific that you can do in the emergency room for this burn except to get the clothing off the patient and get him on sterile sheets. The treatment is so varied in terms of what any individual surgeon prefers, that I cannot give you clear guidelines. Put the patient on sterile sheets, remove the clothing, and insert an I.V. Get two I.V.'s started in any major burn. Get a Foley catheter in. Go by what your local hospital's surgical department has decided as to the way they should treat burns, which will be either by exposure or by the closed method. If by the closed method, you can start your dressings in the emergency room. If it is going to be an exposure situation, put him on the sterile sheets, cover him with sterile sheets, and that is it.

Summary

To sum up then, we have considered a priority order of "things to do" for any patient presented with multiple system injury. Emphasis was placed on immediate assurance of an adequate airway. This is followed by replacement of intravascular catheters for the administration of first saline, followed by whole blood as soon as it is available. Coupled with this is an additional catheter for monitoring the venous pressure. Next, attention is turned to splinting fractures, relieving pain and starting antibiotics, tetanus prevention, and gas gangrene prevention, if indicated.

The various body systems most frequently involved in multiple system injuries were then reviewed in specific order. This was done deliberately in the order presented, to provide you with a systematic scheme in analyzing this type of patient. Because they are so sick and so much must be done rapidly, you must use such a scheme to avoid overlooking one or another possibility.

You will note that little mention has been made of the use of the X-ray department. Almost everything you need to know about these patients for their initial care is readily available to you just for the looking, feeling, listening and probing.

X-ray is a wonderful ancillary tool, but it should be used after all of the initial care has been started and because motion of the patient will upset his hemodynamic balance. It should be kept to a minimum.

The system problems perhaps were outlined less specifically than some of you would like to have considered them, so I will be happy to answer any questions that you have.

CARE OF SIMPLE WOUNDS

Lindley Reagan, M.D.*

A Disaster

The care of simple wounds is important, within the reach of everyone who will take pains. It is something to which you cannot just give lip service. A good example was the hurricane disaster in Worcester about ten years ago that was studied by Dr. John Raker. This whole thing was a disaster - in planning, in management, and in practically every aspect. The storm hit the outskirts of town. About 90 percent of the injuries were taken to small hospitals while the two major hospitals in the city got about three patients each. This meant that they were terribly overburdened outside and paid no attention inside.

The hospitals were flooded. Hospital staffs were unable to handle adequately the hundreds of wounds, but they did their best. Staff went from patient to patient with the same suture set without debriding, without irrigation, without really taking care of them as you should; and in so-doing, they turned simple wounds into very complicated, deep infections which literally took months to clear up. However, there were two neurosurgeons who handled things as they should be handled. They got word to the squads and the accident wards in the other hospitals that all patients with head injuries and head wounds should be sent only to two places. When patients came to the two points of referral, they were treated properly. This was the only group of wounds that were not complicated and did not require many weeks of care. Of course, the patients with head injuries were helped by the fact that, in general, scalp wounds tend to heal kindly.

A History Important

I think that with any medical condition, you should begin with a history if you can. This doesn't mean a long history, but it is a good thing to know how the accident happened. If it is a stab wound that resulted from an intent to hurt, you need to know it. A wound which is one-half inch long in the skin may be six inches long in the liver. On the other hand, the gash which is six inches long on the abdomen may be nothing at all except a flesh wound. If you can, get a history, evaluate it, and take it with a grain of salt. You will get kids coming in with a little scratch on their knuckles or a husband who will tell you that he accidentally cut his hand while using a knife. You don't find out until two days later when it is infected, that he got it by hitting his wife in the teeth. People will try to hide their bad behavior. Certainly there is no wound which is dirtier or more dangerous than a bite wound. It can cripple the hand, if not kill the patient.

Diagnosis

It is also important to make a diagnosis as to what is anatomically wrong in that hand if you can. This also is important in something such as a small wound of the eyelid, particularly if you are in an area where there is a good

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deal of physical combat. A deep eyelid gash, particularly if it goes through the tarsal plate is a disfiguring and potentially disabling injury and it has to be carefully repaired. You have to look for this. An eyelid wound that goes through the plate is no longer a simple wound.

Abdominal Wounds

This is also true of abdominal wounds. Again, particularly there where they are stab wounds, it is not an easy matter to decide what to do. It used to be easy. The bulk of surgical services still have a rule that any stab wound of the abdomen shall have a laparotomy. In many ways this is a safer way to do it. It has built-in hazards however, which are considerable. Somebody with a stomach full of beer and pretzels, and with a stab wound is not a good anaesthetic risk. If you do a laparotomy for, in fact, what is only a flesh wound, you have exposed him to great hazard -- aspiration pneumonia or worse. There is good authority for treating abdominal stab wounds with a certain amount of discretion, judgment, and careful observation.

You need to know what is an abdominal wound, and this can be confusing. We had a patient who had a little knick about half way between the xiphoid and the umbilicus and promptly went into shock. We gave him about four or five pints of blood, trying to resuscitate him. Finally when he was in reasonable shape, we did a laparotomy, only to find absolutely nothing. The abdomen was clean and dry - there wasn't a wound there at all. It wasn't until the next morning that it became apparent even to us that he had a chest full of blood! The knife had gone through the skin and the diaphragm, without wounding anything intra-abdominally. So with abdominal wounds, in general, as emergency room surgeons, you will probably be wise to call for the surgical service to come to help you with them. You do have to recognize that they are problems in order to act. A stab wound of the flank, buttocks, or of the lower chest, may well be an abdominal wound. You have to think about this.

Observe Carefully

The ways to observe simple wounds and diagnose them are getting increasingly sophisticated and gadget-ridden. Even with the new facilities that are available in many places, it is hard to beat just careful observation. One must go back and repeatedly examine the patient, watching for any abdominal tenderness, signs of ileus, increasing leukocytosis, and so on. This is more reliable than use of gadgets alone.

Another method being used more and more is that of sinograms of the wound. A catheter is sewn snugly into the wound and then injected with 40-50 cc of radio-opaque dye. This can reveal puddling in the peritoneal cavity, outline the visceral surfaces of the bowel, and make a very clear diagnosis of abdominal penetration.

Before I leave the abdomen, I think it is totally silly to probe the wound. This makes no sense at all, hurts the patient, and gives you no information which is of value. The fact that you cannot get a Kelly or a blunt instrument into the wound does not mean anything at all.

Tendon Injuries

Tendon injuries are not always easy to diagnose. I think it is a question of how far you should go in trying to prove if there is tendon injury or not. The fact that a patient can bend his fingers doesn't mean that he has not had a major and disabling injury. I am sure you know that the flexor profundus tendons attach only to the distal joint. If the flexor profundus is divided, flexion remains possible at metacarpo-phalangeal and first interphalangeal joint. You have to look with a certain amount of care. As an intern, I called a staff man about 15 miles away at midnight, to help me with a "tendon" injury. The boy patient was cut on the forefinger. He couldn't flex it at all - until he saw the operating room! People with a painful cut often will not do what you ask them to do, and you may be fooled.

There is a great deal to be said, however, for not inspecting these wounds too much. You can convert a clean wound into a dirty wound. It is still true, unfortunately, that hospitals can be very dangerous places to have people tamper with wounds. Instruments can be infected with pathogenic organisms that are not just ordinary bugs, and a dangerous infection results. It goes without saying that all flexor tendons of the hand are extremely complicated. Flexor tendons should go to the operating room for expert care. Almost never should they be sutured in an accident ward. Again this has to be tempered by judgment. I see no reason why a partially severed tendon, if conditions are right, and you handle the wound carefully, cannot have one or two coaptation sutures put in it. I don't think you will get damage if you are clean in your preparation.

It still remains true that the safest wound is an unsutured wound. A wound that is poorly handled and closed is far more likely to get infected than the wound which is simply left open without a bandage on it. It would not be proper to leave a wound unbandaged indefinitely, but as military surgeons have shown over and over there is maximum safety in the wound which is left open.

Nerve Injury

You must look for evidence of nerve injury and this can be difficult in somebody who has had sixteen cocktails or the ordinary "two beers" before he came in. Although the patient doesn't know whether he can feel pinprick or not, you should try. It is very frustrating to be in the operating room trying to decide whether it is a nerve injury or not, with the patient asleep. There is no way of knowing unless you really dissect that nerve, and this is often difficult to do. If you can prove before patients go to sleep that they do or do not have nerve injury, you are much more comfortable in the operating room with them. There are simple tests - the finger pad of the little finger is a pure ulnar nerve innervation, and the finger pad of the forefinger is a pure median nerve innervation - and if you get anaesthesia into those two places you can locate the nerve injury exactly. This is about as simple a way to tell as there is. The radial nerve does not give an anaesthesia pattern. A little hypaesthesia in the thumb web space may be the only sign. With wounds higher on the arm, you can be fooled with the radial nerve, because there is a difference between wrist drop and finger drop; both are mediated by the radial nerve. An injury proximal to the elbow can give a total radial palsy, in which there will be wrist drop and

finger drop. An injury in the area of the outer external condyle may allow dorsiflexion of the wrist but not of the fingers. Thus you can have wrist extension, but still have a disabling radial nerve injury.

Arterial Injuries

There has been a lot written about arterial injuries and about how subtle they can be. They can be subtle and lethal, at least to a limb, if not the patient. You can be fooled because pulses can be transmitted across a partially lacerated artery. One should think about arterial injury, if the wounds are in places where they can involve vessels as in the elbow, the groin, and neck. More and more hospitals are equipped now as do emergency arteriograms which often are indispensable.

On handling the wound itself, I might appear again to be quoting the primer to you, but the Worcester disaster showed that this is important. The basic concepts of wound care are things that everybody knows, but in general, everybody ignores to a greater or lesser extent. There is no substitute for (A) being gentle with the wound, and (B) there is absolutely no substitute for cleaning the wound. The time to treat an infection is before it starts, and not afterwards. Once the bugs get invasive and growing, the damage is done. If you can keep bugs from getting into wounds, or keep them from staying there, then you have made a real contribution to the patient's care. Wounds have to be thoroughly cleansed and irrigated. This will save time. It may not save time in the immediate care of the wound, but it will save weeks of redressing and crippling. It requires a lot of water, lots of time, and anesthesia.

Anesthesia

These are emergency patients and anesthesia is a problem. Hardly anybody dies of a cut hand, but people die if they inhale a chicken sandwich. If possible, general anesthesia should not be given to these persons. It is old hat for me to tell you that digestion stops at the time of injury. If they are hurting, nothing is going on in their stomach. Everything they ate hours ago is just sitting there waiting to be aspirated. You should use general anesthesia as sparingly as you can, and almost the only times I think you are obligated to use it, is for dislocation and hemorrhage. Dislocations should be treated as soon as possible and without waiting. Most other things can be delayed. We have been using a lot of IV Xylocaine blocks. This has solved a lot of problems for us, and it is ideal anesthesia for the injured hand and forearm. This gives what amounts to general anesthesia of the extremity. It is simple, but hazardous if it is not done as directed in the book. When this was first used, there were fatalities with cardiac arrest. We use 0.15 cc/lb. of one-half of one percent Xylocaine. This is safe. This is done by placing a tourniquet and putting a scalp vein needle in the vein, but not injecting the Lidocaine yet. Then the extremity is made bloodless by an ace bandage wrapped tightly from the distal extremity on up to the pneumatic tourniquet or pressure cuff. Once wrapped to that point, the tourniquet is inflated to 250 mm. of mercury and clamped. The ace bandage is then removed and the extremity is relatively bloodless. Inject the Lidocaine in the previously placed IV needle. Anesthesia requires 20-40 minutes. The biggest trouble with local anesthesia in most people's hands is that they do

not wait long enough. It makes the arm look awful, but this is good anesthesia for lacerations of two or three fingers of a simple Colles fracture of the wrist or something of that sort. You can keep this in place for up to two hours, and it will give good anesthesia. If the tourniquet gets loosened or if the clamps come off, then anesthesia is gone almost instantly.

Debridement

Debridement should be thorough within reason. Debridement of wounds of the face should be conservative. Saw-tooth and irregular edges should be converted to straight ones; devitalized tissue ought to be removed. Remember that the blood supply of the face is extremely rich and you do not have to remove as much tissue as you would other places. You have to decide whether you should suture this wound, or whether you should in fact leave it open. Many dirty wounds should be treated by cleaning them up as best you can, and leaving them open. Patients can be brought back, preferably within three or four days, and the wound revised and closed. Normal saline is the best irrigant to use. We use a combination of this and PhisoHex.

On how long you leave the sutures in, you quickly learn when you have been wrong. It is quite irritating to have somebody call you at midnight to say that the cut popped open. The old rule is true that a wound heals faster, the higher the temperature. It is another way of saying that wounds on the face and head heal quicker than wounds on the fingers, the back and the feet. It also depends upon how the wound is oriented. If you can plan an operative incision, this is one thing; but if it crosses the elastic lines, and so on, you must leave the sutures in longer. I think a face wound which is well approximated and in proper lines, can have the sutures out in three or four days. If not so oriented they may require up to a week. It is nice to get the sutures out on the face as quickly as you can, so that you don't leave suture marks. On the back, on the extremities, and the hands, the sutures should be left almost a minimum of two week's time. Otherwise some of them will split.

Sutures

I think there is so much discussion and controversy over suture material to use that I think that it is largely fielder's choice and what you are used to. In our Accident Ward, we use wire almost exclusively. I think the fact remains that wire is the best tolerated, least irritating suture material that there is beyond any question; monofilament, single strand wire, not the braided wire. In a badly contaminated wound, wire is the suture material of choice. It is uncomfortable and often hard to get out. I often use just plain cotton or silk sutures of small calibre, the finest that you can use. It is most distressing to see a little wound come in with 0 or 1 silk in it. This is the kind of stuff you use to anchor an ocean liner. You don't need it. Sutures need to just be strong enough to hold the tissues which they are joining. A wound of the face, the eyelids, ear requires 5-0 or 6-0 material. The newer synthetic sutures are all right, although a reliable knot requires 4-6 throws. For tissues which are very thin, very pliable, it is better to use a pliable suture, and to me this means silk. Use buried sutures sparingly. They should be fine catgut, 4-0 or 5-0. On a face wound which is in the wrong direction for elastic fibers, it is

good to put in some catgut to prevent spreading. If the skin sutures are left long enough to prevent spreading, permanent suture marks are made. With these wounds it is often well to build them up with cutaneous fine catgut.

Tetanus

Tetanus toxoid and anti-tetanus measures are always important. It is much simpler now that human anti-tetanus serum is available. You must remember that tetanus toxoid is absolutely of no value for a present wound. It can give no immunity for a period of some several weeks to the person never immunized. For anyone properly immunized, however, it is probably safe forever. Certainly this is still true of World War II veterans. These men, if given a tetanus booster, will within a period of hours have a satisfactory level. Most school children have also had it. There is no excuse any more for using TAT. It is unreliable; it doesn't last very long, and gives an appreciable element of serum sickness which can be quite miserable if not down right dangerous. Sensitivity to horse serum can be almost instantly fatal. Now-a-days with this human anti-tet, there is no reason for using TAT. Encourage the patient to have boosters and having this, they are safe for a long period - at least 5 years. The business of giving everybody who comes in a repeat booster of toxoid no matter when they had the last one, is needless and potentially harmful. You can certainly wait three years before giving another booster. If patients don't remember and their parents have forgotten, then it is better to use it than to make a mistake. If it is over three years, you should give a booster. The last thing I read was that it was suggested that a booster be given every ten years.

Puncture Wounds

I think you have to be careful about puncture wounds and blast wounds of one sort or another, because they can be treacherous. We have seen a 12 year old girl come in with a wound about 1" long on the leg, allegedly from a stone thrown out by a rotary mower. X-rays revealed a 6" screw driver in the leg! When there is a question of a blast or something being thrown, you have to X-ray it, no matter what the patient tells you. This is true of firecrackers or of explosives where a good deal of wadding or cardboard material can be driven in. It is true in penetrating wounds of the feet where a piece of shoe leather can often be thrown in, and this will often show on X-rays. You should routinely X-ray this kind of wound.

Foreign Bodies

On foreign bodies: leave them alone! The only time a foreign body should be removed in the Accident Ward is when it can actually be felt before you start. These should be done with full anesthesia and a tourniquet in place. There is nothing that can be more exasperating than a foreign body even if it looks very simple on X-ray. You need all the help you can get. There is no reason why you cannot wait until the next morning to remove it in the operating room under good conditions, with the patient fasting.

Infections

I must add a word about people with infections. I have seen too many people who have infections which have been treated with antibiotics. Antibiotics will not cure pus, no matter what. You cannot wait for the ordinary signs of fluctuation in some areas. If you wait until a felon is fluctuant, you have destroyed the distal phalanx. If somebody has been kept awake all night with pain, this is almost reason enough to incise and drain right then. The same thing is true of many deep peri-anal abscesses. You will see people in real pain for three or four days, getting antibiotics. This is wrong. Generally if there is considerable pain, there is a collection which can be found, which I think should get surgical action forthwith, usually with general anesthesia.

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